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CONTENTS

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BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST

Vol. XLVII. JANUARY, 1953 No. 1.

EDITORIAL

THIS number of the BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST marks the opening of its forty-seventh year, and we feel that we should use this Editorial as an opportunity for stocktaking and reflection.

For twelve months now the Journal has been considerably increased in size, in line with the growing importance of the study of diseases of the chest. This expansion is reflected also in the more numerous contributions which we are receiving not only from this country and the Dominions but from other countries as well. This happy state of affairs is in no small measure due to the invaluable help given by the Editorial Board in Britain and the Dominions, who keep our readers informed of developments in their respective countries.

We can contemplate with pride the astonishing advances in the diagnosis and treatment of diseases of the chest which compare favourably with those in any other department of medicine or surgery. Because of advances in chemotherapy and thoracic surgery it is not surprising that the main emphasis, for example in tuberculosis, has been on treatment of the established case, although these advances must be recognised as ancillary aids and not as substitutes for certain well-established and fundamental principles which have stood the test of time.

We must ask ourselves whether more thought and activity might not now be given to new methods of prevention and control, pursuing the advantages obtained from mass radiography, tuberculin surveys, contact examinations, B.C.G. vaccination and the like. Other promising studies in the field of prevention have been made by Cochrane, Cox, Glyn and Jarman,[1] who investigated a complete community in a South Wales mining valley, and by Stewart and Hughes[2] on "Infection in Factories." Further studies on the epidemiology of the disease should yield fruitful results, and a contribution by B. Benjamin on "Tuberculosis and Social Conditions in the Metropolitan Boroughs of London" in our present issue is a case in point.

In the words of the late J. A. Ryle,[3] ought we not to assume a "new, broader and more intellectually inspired sense of duty towards our social organisation and our social needs and hazards"? Social pathology in relation to etiology and prevention should be no less important than individual pathology in relation to diagnosis and treatment.

In relation to modern developments there will, we think, be general agreement with B. A. Dormer's view[4] of the new outlook wherein the "Tuberculosis Officer" of the past, specialising in tuberculosis, has been replaced by the "chest physician" who, prior to specialisation in tuberculosis and diseases of the chest, has been well trained in general medicine.

Specialists in every field must formulate for themselves a general philosophy of medicine in the light of which they will be able to assess the position of their own speciality. Common to all is the danger of compartmentalism, against which Professor Sir Henry Cohen[4] has uttered words of warning " that the methods of history-taking and of clinical examination are the same whatever the site and nature of the disease, and the practice of medicine demands that man must be treated as a whole." Here we would add that such a history must probe beyond the clinical picture to the social, dietetic, psychiatric, occupational and other factors which may have bearing on the causation or the course of the disease.

We must ask ourselves, too, what are the limits of our speciality and how restricted is its field of vision to be. Can the heart and lungs, for example, lying within the thoracic cage, continue in isolation? The close anatomical and functional relationship of these organs, which work as a single oxygenating unit, indicates that disorders of deranged function in the one may easily lead to secondary changes in the other, and that damage to any part of the circuit will affect the efficiency of the whole.

The chest physician is constantly encountering symptoms such as dyspnœa, cough and hæmoptysis common to heart as well as lung disease. For his correct assessment he will fall back on his fundamental training in general medicine.

At this juncture it is not without interest to refer to a leading article in the *Lancet* on " Trends in Cardiology."[5] With reference to the first Congress of Cardiology held in London last September it says: " The meeting provided an opportunity for appraising the position of this speciality in relation to the whole fabric of internal medicine." The Congress had made it clear that cardiology retained its place as one of the most scientific of the divisions of medicine. The *Lancet* continues: " Judging by the papers at the Congress, cardiology is more concerned with ' ends ' than ' beginnings ' of disease." It goes on to deplore the almost total absence of etiology from the discussions. Moreover, it continues: " It is only in recent years—and largely because of successful surgical intervention in cardiac disease—that cardiologists and specialists in pulmonary disease have taken notice of each other. They have long lived in the same house, hardly recognising each other's existence. Now, however, the chest physician appreciates that the pulmonary circulation is of fundamental importance to the efficiency of the lungs, while the cardiologist acknowledges that the physical environment of the blood in its passage from pulmonary valve to left auricle is a major factor in cardiac function."

A sense of proportion and the exercise of sound judgment are of the first importance in prevention and treatment of disease whose social and economic aspects weigh almost equally with the medical factors. In safeguarding and improving the quality of the work in our tuberculosis and chest services we should do well to remember the counsel of Dr. B. A. Dormer,[6] the South African member of our Editorial Board, in his article which appeared in our January issue of last year.

As far as the future of this Journal is concerned, we shall continue to publish original work in tuberculosis, diseases of the chest and cognate subjects, more particularly in the preventive and therapeutic spheres. Signed book reviews giving, as Hugh Clegg[7] points out, a heightened and not a diminished

sense of responsibility to the author of the book have been a new feature of the last year. We shall endeavour to maintain in 1953 the present standard of the Journal, recognising with Professor Leslie Banks that " if the practice of medicine is to be kept at an economic level and at the same time incorporate all the benefits of present progress, the medical profession must pay more than lip service to the adage that prevention is better than cure, for it applies to the health of the medical profession as well as to the health of the nation."

Let us hope that future trends will not regard the preventive and curative aspects as distinct entities and that, as Banks rightly urges, prevention, diagnosis, treatment and after-care will be established as a continuous process, rather than proceed on the present narrow lines of specialisation.

P. E.

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GENERAL ARTICLES

TUBERCULOSIS AND SOCIAL CONDITIONS IN THE METROPOLITAN BOROUGHS OF LONDON

By B. Benjamin

Formerly Statistician, London County Council.*
Now Statistician, General Register Office

Introduction

This survey attempts to compare the incidence and trends of pulmonary tuberculosis in each of the metropolitan boroughs, excluding the City of London which is too small to be of statistical value. It was thought that such a comparison (between areas with broadly uniform clinical services) was not only a normal requirement of tuberculosis control in London but that it might provide valuable information as to the relative importance of social and environmental factors to the extent to which the variation of these factors among the Boroughs might be measurable.

Certain reservations must be mentioned. Whilst the tuberculosis mortality statistics of any area are reliable, the only measure of morbidity available is the rate of notification under the Public Health Act, 1936. At all times, notification is naturally subject to a degree of incompleteness, however small (Stocks, 1949), which may vary locally.

A second difficulty is that although the metropolitan boroughs may be graded by indices of housing density or extent of employment in lower paid occupations, etc., these indices represent broad averages and the conditions described are by no means *uniform* over an individual borough. Considerable heterogeneity in fact exists.

The third difficulty arises from the fact that every day some three-quarters of a million persons from the dormitory areas surrounding the metropolis come into that area to work, while there is a reverse movement of a fifth of a million. Inside London there is a daily movement of nearly a million workers. The geographical spread of this daily movement is not uniform (Menzler, 1951) and tends to become concentrated toward commercial areas of the centre and other more industrialised parts of the County; it must not only have an influence on the spread of tubercle but must tend also to blur variation from borough to borough.

Finally, towards the end of the period—namely, in the war and post-war years, from about 1942—a substantial part of the rise in the notification rates is due to an increased intensity of case-finding, which is not necessarily equally incident on all boroughs.

* This study was made while the author was Statistician to the London County Council Health Department.

(*Received for publication June* 11, 1952.)

The Trend 1931-48

Table I and Table II show the mortality and morbidity rates* in the London boroughs per 1,000 of the population.

Table I.—Death Rates from Pulmonary Tuberculosis per 1,000 Living

	Population (Census 1931)	1931-33	1934-36	1937-39	1940-42	1943-45	1946-48	Population (Mid-year 1948)
Division 1:								
Chelsea	59,031	0·76	0·72	0·75	0·94	0·82	0·45	50,890
Fulham	150,928	0·92	0·77	0·77	0·85	0·79	0·57	123,300
Hammersmith	135,523	0·95	0·84	0·72	1·05	0·81	0·64	115,800
Kensington	180,677	0·65	0·54	0·57	0·72	0·73	0·45	168,800
Division 2:								
Hampstead	88,947	0·41	0·42	0·39	0·63	0·60	0·35	95,480
Paddington	144,923	0·72	0·54	0·61	0·88	0·74	0·63	128,100
St. Marylebone	97,627	0·71	0·67	0·54	0·77	0·65	0·46	75,980
St. Pancras	198,133	0·90	0·78	0·73	1·14	1·05	0·68	140,200
Westminster, City of	129,579	0·76	0·61	0·63	0·86	0·87	0·61	100,900
Division 3:								
Finsbury	69,888	1·13	0·84	0·78	1·50	1·22	0·87	35,640
Holborn	38,860	0·84	0·83	0·71	1·22	0·87	0·60	24,960
Islington	321,795	0·87	0·72	0·75	0·98	0·84	0·67	238,200
Division 4:								
Hackney	215,333	0·74	0·64	0·56	0·78	0·69	0·55	172,900
Shoreditch	97,042	0·93	0·75	0·80	1·53	1·00	0·79	44,960
Stoke Newington	51,208	0·86	0·70	0·58	0·86	0·61	0·50	45,370
Division 5:								
Bethnal Green	108,194	0·91	0·66	0·70	1·08	0·82	0·72	60,580
City of London	10,999	0·68	0·71	0·65	0·66	0·59	0·67	4,810
Poplar	155,089	0·92	0·76	0·75	1·24	1·07	0·85	74,940
Stepney	225,238	0·94	0·75	0·69	1·19	1·28	0·78	99,470
Division 6:								
Deptford	106,891	0·91	0·80	0·65	0·94	0·86	0·55	75,670
Greenwich	100,924	0·90	0·72	0·69	0·96	0·77	0·74	84,410
Woolwich	146,881	0·91	0·79	0·72	0·91	0·79	0·56	142,800
Division 7:								
Camberwell	251,294	0·91	0·76	0·72	0·93	0·84	0·60	178,200
Lewisham	219,953	0·63	0·60	0·63	0·76	0·75	0·52	226,100
Division 8:								
Bermondsey	111,542	1·01	0·88	0·75	1·04	0·98	0·73	60,410
Lambeth	296,147	0·85	0·74	0·75	0·98	0·92	0·62	226,600
Southwark	171,695	1·08	0·85	0·82	1·23	1·39	0·83	94,630
Division 9:								
Battersea	159,552	0·87	0·78	0·72	0·99	0·83	0·62	116,500
Wandsworth	353,110	0·74	0·64	0·59	0·82	0·66	0·52	332,500
London County	4,397,003	0·84	0·71	0·68	0·95	0·83	0·60	3,339,100

* The rates for war and post-war years are based upon civil populations, since only these populations were available; some inflation of the rates is an inevitable consequence, the fitter members of the general population being excluded by recruitment to the Forces. The denominator of the rate is reduced without any corresponding reduction of the numerator. Whilst this has the effect of introducing a false hump in the secular trend in 1940, it is a feature common to all boroughs and therefore does not invalidate inter-borough comparisons.

It was clear from statistical tests of homogeneity that for the purpose of typifying the London boroughs the average County rate was useless—the boroughs did not belong to the same family of tuberculosis experiences.

TABLE II.—PRIMARY NOTIFICATIONS OF PULMONARY TUBERCULOSIS PER 1,000 LIVING

	Population (Census 1931)	1931-33	1934-36	1937-39	1940-42	1943-45	1946-48	*Population (Mid-year 1948)*
Division 1:								
Chelsea	59,031	1·55	1·43	1·32	1·66	2·07	1·45	50,890
Fulham	150,928	1·62	1·51	1·48	1·84	2·04	1·79	123,300
Hammersmith ..	135,523	1·38	1·32	1·25	1·73	2·08	1·58	115,800
Kensington	180,577	1·10	0·96	1·03	1·69	1·63	1·32	168,800
Division 2:								
Hampstead ..	88,947	0·97	0·76	0·87	1·39	1·57	0·98	95,480
Paddington	144,923	1·45	1·31	1·53	2·11	2·42	2·25	128,100
St. Marylebone ..	97,627	1·50	1·20	1·19	1·82	1·85	1·20	75,980
St. Pancras	198,133	1·34	1·24	1·35	1·83	2·18	1·69	140,200
Westminster, City of	129,579	1·42	1·49	1·41	1·93	2·35	1·78	100,900
Division 3:								
Finsbury	69,888	1·81	1·52	1·39	2·25	2·37	1·80	35,640
Holborn	38,860	1·36	1·69	1·64	2·27	2·35	2·03	24,960
Islington	321,795	1·50	1·48	1·57	1·84	2·20	1·77	238,200
Division 4:								
Hackney	215,333	1·25	1·13	1·12	1·50	1·67	1·41	172,900
Shoreditch	97,042	1·54	1·27	1·20	1·96	2·05	1·66	44,960
Stoke Newington ..	51,208	1·29	1·18	0·83	1·22	1·59	1·56	45,370
Division 5:								
Bethnal Green ..	108,194	1·17	1·17	1·14	1·54	2·00	1·63	60,580
City of London ..	10,999	2·12	1·97	1·56	1·98	2·78	2·13	4,810
Poplar	155,089	1·41	1·25	1·15	1·68	2·13	1·70	74,940
Stepney	225,238	1·72	1·57	1·27	2·04	2·43	1·84	99,470
Division 6:								
Deptford	106,891	1·54	1·31	1·39	2·04	2·09	1·51	75,670
Greenwich	100,924	1·14	1·15	1·15	1·57	1·63	1·38	84,410
Woolwich	146,881	1·52	1·46	1·63	1·88	2·02	1·63	142,800
Division 7:								
Camberwell ..	251,294	1·26	1·16	1·28	1·60	1·81	1·93	178,200
Lewisham	219,953	1·27	1·29	1·28	1·51	1·90	2·00	226,100
Division 8:								
Bermondsey ..	111,542	1·63	1·34	1·23	1·89	2·52	1·90	60,410
Lambeth	296,147	1·58	1·39	1·39	1·73	2·00	1·53	226,600
Southwark	171,695	1·61	1·66	1·66	2·47	2·66	2·13	94,630
Division 9:								
Battersea	159,552	1·28	1·35	1·29	1·70	2·04	1·54	116,500
Wandsworth ..	353,110	1·22	1·13	0·94	1·45	1·59	1·36	332,500
London County ..	4,397,003	1·40	1·31	1·29	1·74	1·98	1·64	3,339,100

There were differences of overall level or of trend or both. One of the principal objects was to separate these differences. The County average, though unrepresentative, was used (in the rôle of a purely notional trend) as a yardstick by which to group the boroughs as follows (where the terms " fixed," " increasing," " decreasing " apply only approximately):

COMPARISON WITH COUNTY AVERAGE (* INDICATES STATISTICALLY SIGNIFICANT DEVIATION)

	A. Mortality		*B. Morbidity*	
1. Higher by increasing amount:				
	St. Pancras*	Poplar*	Paddington*	Bermondsey*
	Shoreditch*	Stepney*		
2. Higher by fixed amount:				
	Finsbury*	Holborn	Westminster*	Islington*
	Southwark*	Bethnal Green	Southwark*	Holborn*
	Lambeth*	Battersea	Deptford*	Hammersmith
	Islington	Greenwich		
3. Higher by decreasing amount:				
	Hammersmith*	Bermondsey*	Fulham*	Finsbury*
			Stepney*	Woolwich*
4. Higher at first then lower:				
	Fulham	Deptford	Lambeth*	Chelsea
	Camberwell	Woolwich		
5. Lower then higher:				
	Chelsea	Westminster	St. Pancras*	Poplar
			Shoreditch*	St. Marylebone
6. Lower by decreasing amount:				
	Paddington*	Hampstead*	Kensington*	Lewisham*
	Kensington*	Lewisham*	Bethnal Green*	Camberwell*
7. Lower by fixed amount:				
	St. Marylebone*	Wandsworth*	Hampstead*	Greenwich*
	Hackney*		Stoke Newington*	Battersea
			Hackney*	
8. Lower by increasing amount:				
	Stoke Newington		Wandsworth*	

These group averages were then plotted in Diagram A.

DIFFERENCES IN BOROUGH TRENDS

(i) *Mortality*

There is no area in which some decline in tuberculosis mortality has not been taking place. Some areas such as in group A.1 in the above classification (St. Pancras, Shoreditch, Poplar, Stepney) appeared to be behind the general rate of progress though not lacking in overall improvement. These are, in in the main, inner areas of higher than average industrialisation (especially in occupations known to have a high tuberculosis mortality risk (*m*), Table IV); in 1931 they were areas with high percentages of the working populations in the Registrar General's social classes IV and V (see column (*b*), Table IV), higher housing density (*c*, Table IV) and subject to adverse selection in many other social factors. Generally speaking, the areas of high tuberculosis mortality prior to the 1939-45 war were those which also suffered the more severe upward fluctuations in mortality during the war. These upward fluctuations were exaggerated by war-time evacuation which tended to exclude the tuberculous. Thus the tuberculosis deaths for the area were not depleted in the same proportion as the population (Benjamin and Daley, 1942). Similar remarks can be applied to Group A.2. Groups A.3 and A.4 (Hammer-

smith, Bermondsey, Fulham, Camberwell, Deptford, Woolwich), though originally higher than the County average, appear to have made more than average progress and to have improved their relative position. In Hammersmith, Fulham and Camberwell the economic circumstances were above average, while in Bermondsey, Deptford and Woolwich, where poorer conditions obtained, expenditure on tuberculosis services per case was above average and considerable effort seems to have been made. On the other hand, a few boroughs of good social conditions (Groups A.5 and A.6, Chelsea, Westminster, Paddington, Hampstead, Kensington, Lewisham), though experiencing lower than average mortality, have not maintained the average rate of progress. It may well be that in the general movement of the County of London there has been selective migration of the healthier elements from these areas.

TABLE III.—RATIO OF DEATHS TO PRIMARY NOTIFICATIONS

	1931-33	1934-36	1937-39	1940-42	1943-45	1946-48
Division 1:						
Chelsea	0·49	0·50	0·57	0·57	0·40	0·31
Fulham	0·57	0·51	0·52	0·46	0·39	0·32
Hammersmith	0·69	0·64	0·58	0·61	0·39	0·41
Kensington	0·59	0·56	0·55	0·43	0·45	0·34
Division 2:						
Hampstead	0·42	0·55	0·45	0·45	0·38	0·36
Paddington	0·50	0·41	0·40	0·42	0·31	0·28
St. Marylebone	0·47	0·56	0·45	0·42	0·35	0·28
St. Pancras	0·67	0·63	0·54	0·62	0·48	0·40
Westminster, City of	0·54	0·41	0·45	0·45	0·37	0·34
Division 3:						
Finsbury	0·62	0·55	0·56	0·67	0·51	0·48
Holborn	0·62	0·49	0·43	0·54	0·37	0·30
Islington	0·58	0·49	0·48	0·53	0·38	0·38
Division 4:						
Hackney	0·59	0·57	0·50	0·52	0·41	0·39
Shoreditch	0·60	0·59	0·67	0·78	0·49	0·48
Stoke Newington	0·67	0·59	0·32	0·70	0·38	0·32
Division 5:						
Bethnal Green	0·78	0·56	0·61	0·70	0·41	0·44
City of London	0·32	0·36	0·42	0·33	0·21	0·31
Poplar	0·65	0·61	0·65	0·74	0·50	0·50
Stepney	0·55	0·48	0·54	0·58	0·53	0·42
Division 6:						
Deptford	0·72	0·61	0·47	0·46	0·41	0·36
Greenwich	0·79	0·63	0·60	0·61	0·47	0·54
Woolwich	0·60	0·54	0·44	0·48	0·39	0·34
Division 7:						
Camberwell	0·72	0·66	0·56	0·58	0·46	0·31
Lewisham	0·40	0·47	0·49	0·50	0·39	0·26
Division 8:						
Bermondsey	0·62	0·66	0·61	0·55	0·39	0·38
Lambeth	0·54	0·53	0·54	0·57	0·46	0·41
Southwark	0·67	0·51	0·49	0·50	0·52	0·39
Division 9:						
Battersea	0·68	0·58	0·56	0·58	0·41	0·30
Wandsworth	0·61	0·57	0·63	0·57	0·42	0·38
London County	0·60	0·54	0·53	0·55	0·42	0·37

Groups A.7 and A.8 have had very favourable trends. They have economic advantages in social class and housing which have been maintained.

(ii) *Morbidity*

Post-war morbidity rates are not comparable, for reasons already given, with pre-war rates. The present discussion is therefore confined to the 1931-39 period. Before the war even in areas of high morbidity the notification rate was declining with the exception of Paddington, St. Pancras, Kensington, Hampstead, Camberwell, Deptford and Islington, where, in the immediate pre-war years, there was a tendency for the incidence of pulmonary tuberculosis to rise. In certain areas such as Fulham, Finsbury, Stepney, Lambeth and Chelsea there was a suggestion that the rate of occurrence of new cases was falling, if only slightly, relative to the County average. Kensington and Lewisham, again, receive mention as boroughs which, though areas of low notification rate, suffered some deterioration in their position relative to other boroughs, and Bethnal Green and Camberwell must be included with them. On the other hand, Wandsworth increased its advantage.

(iii) *Ratio of deaths to cases*

It will be clear from the above statement that the position of any borough relative to the others varies according to whether the mortality rate or the notification rate is considered. It is to be expected that where the intensity of case-finding is high the cases would be found early and the prognoses would be more favourable, so that a high notification rate might be associated with a low mortality rate. On the other hand, in an area where tubercle was rife the notification rate might still be high without intense search for cases, but mortality would also be high.

In Table III the ratio of deaths to cases notified is shown for each borough for the same triennia as in Tables I and II. The boroughs may then be grouped according to the following plan:

Tuberculosis Death Rate (Deaths Related to Population)	*Case Fatality—i.e., Ratio of Deaths to Cases*	*Boroughs*	
High ..	High ..	Hammersmith	Poplar
		St. Pancras	Southwark
	Average ..	Finsbury	Bermondsey
		Shoreditch	
	Low	Stepney	Lambeth
Average ..	High	Stoke Newington	Battersea
		Bethnal Green	Camberwell
		Greenwich	
	Average ..	Holborn	Deptford
		Islington	Woolwich
	Low	Fulham	Westminster
		Chelsea	
Low	High	—	
	Average ..	Wandsworth	Hackney
		Kensington	
	Low	Paddington	Hampstead
		St. Marylebone	Lewisham

There are no areas, where detection is late in the sense that a high case fatality occurs in conditions of low incidence, but in some areas the number of notifications is not as large as would be expected from the death rates.

Tuberculosis and Social Conditions

An attempt was made to find any association between pulmonary tuberculosis mortality and morbidity (as measured by the primary notification rate of new cases) and other indices which measured the social and economic conditions in the twenty-eight Metropolitan Boroughs, together with indices bearing on the medical services in the period 1931-33, for which comprehensive data were available.

Table IV.—Pulmonary Tuberculosis Mortality and Morbidity Rates and Certain Social and Economic and other Indices for the Metropolitan Boroughs, 1931-33

	z	*a*	*b*	*c*	*d*	*f*	*g*	*h*	*m*	*p*	*o*
Battersea	0·87	1·28	36·1	27·1	11·0	99·1	3·8	4·15	8·0	8·8	25·3
Bermondsey	1·01	1·63	54·7	42·4	14·9	99·6	4·5	3·56	18·9	6·8	17·0
Bethnal Green ..	0·91	1·17	39·9	46·9	15·8	100·3	6·1	2·38	17·6	3·1	15·3
Camberwell	0·91	1·26	33·0	25·2	10·3	100·8	3·1	2·16	8·9	7·6	16·9
Chelsea	0·76	1·55	32·2	25·3	11·1	99·6	8·7	4·72	9·5	33·5	21·3
Deptford	0·91	1·54	41·1	26·2	13·2	100.0	4·6	5·42	9·3	8·2	5·5
Finsbury	1·13	1·81	45·4	52·3	15·5	98·6	6·0	2·75	15·9	9·4	3·9
Fulham	0·92	1·62	28·7	26·3	10·7	99·8	4·0	4·18	8·0	14·6	18·4
Greenwich	0·90	1·14	42·2	25·9	11·4	100·5	4·0	4·00	5·1	11·5	28·1
Hackney	0·74	1·25	26·1	24·7	11·7	100·6	5·3	3·05	10·2	6·1	22·3
Hammersmith ..	0·95	1·38	31·4	27·6	11·4	99·3	3·6	2·69	7·8	19·9	14·4
Hampstead	0·41	0·97	15·5	12·5	7·1	103·3	4·3	7·36	4·0	29·8	17·5
Holborn	0·84	1·36	29·0	36·9	11·4	100·7	5·2	3·42	14·6	30·0	9·9
Islington	0·87	1·50	32·9	34·3	12·3	98·7	5·5	2·39	10·2	11·1	3·5
Kensington ..	0·65	1·10	30·1	26·6	10·2	100·8	2·9	2·69	8·0	35·2	8·7
Lambeth	0·85	1·58	31·0	25·5	11·1	100·4	4·4	2·82	9·9	12·7	11·6
Lewisham	0·63	1·27	22·7	15·0	6·8	99·3	2·7	2·47	5·1	8·9	18·1
Paddington	0·72	1·45	28·0	27·3	11·1	99·3	8·9	2·35	7·6	29·9	13·3
Poplar	0·92	1·41	61·1	41·9	18·9	98·5	6·2	5·65	12·7	5·7	18·2
St. Marylebone ..	0·71	1·50	27·8	26·9	11·9	102·5	4·6	3·95	9·8	33·5	29·3
St. Pancras	0·90	1·34	33·5	36·9	13·1	99·0	3·7	3·34	12·0	19·4	21·9
Shoreditch	0·93	1·54	44·6	53·0	17·2	97·1	5·5	2·72	18·7	4·7	4·4
Southwark	1·08	1·61	47·0	43·5	14·4	99·6	4·6	3·80	16·4	9·2	7·5
Stepney	0·94	1·72	37·8	46·0	14·4	99·8	3·9	4·10	15·9	5·9	11·5
Stoke Newington ..	0·86	1·29	19·6	18·3	10·3	103·5	5·2	3·75	7·2	9·8	8·0
Wandsworth ..	0·74	1·22	22·3	14·9	8·3	100·8	3·6	2·75	5·5	13·8	19·0
Westminster	0·76	1·42	29·3	21·1	9·7	101·4	3·5	3·18	11·1	38·2	36·2
Woolwich	0·91	1·52	39·3	17·3	9·5	101·3	5·1	4·04	4·8	18·5	29·3
London Average ..	0·84	1·40	33·7	30·3	11·9	100·0	4·8	3·32	10·4	14·5	19·0

N.B.—For the explanation of the code letters (*a*, *b*, *c*, etc.) see the text.

The data used (Table IV) were as follows:

Index

z Death rate from pulmonary tuberculosis per 1,000 of population. Mean annual rate for 1931-33, computed from data provided by the Registrar General.

a Primary notification rate of new cases of pulmonary tuberculosis per 1,000 living. Mean annual rate for 1931-33. Computed from returns made to the L.C.C. by the Borough Medical Officers of Health under the Public Health (Tuberculosis) Regulations, 1930.

b Social Index. Percentage of males aged 14 and over whose occupations were assigned to social classes IV and V at the 1931 Census. Computed according to the procedure given in the Registrar General's Statistical Review for 1934, Text, pp. 150 *et seq.*

c Percentage of population in private families living at a density of more than one and a half persons per room at 1931 Census. Extracted from Housing Report and Tables (Table 13).

d Percentage of males aged 14 and over unemployed at 1931 Census. Extracted from Occupation Tables (Table 16).

f Mean weights of school children expressed as a percentage of the London average, 1938. Computed from L.C.C. Report on Average Heights and Weights of School Children (No. 3464), Appendix I, pp. 10 *et seq.*

g Attendances at tuberculosis dispensaries per case on the registers, 1932. Extracted from Annual Report of the L.C.C., 1932, Vol. III, Part I, p. 36.

h Gross expenditure on the tuberculosis dispensary service in £s per case on the dispensary register in each borough in 1931; figures provided privately by the Comptroller of the London County Council.

m Percentage of occupied males aged 14 and over, engaged at the 1931 Census in the twelve occupations which had the highest mortality from pulmonary tuberculosis during 1930-32. Computed from Table 16 of the 1931 Census Occupation Tables, and Table R and Appendix C of the Registrar General's Decennial Supplement, 1931, Part IIa.

p Proportion of persons per 1,000 total population whose birthplace was shown as Ireland at the 1931 Census. Computed from Table 30, 1931 Census, General Tables, pp. 197 *et seq.*

o Major public open space and private open space, allotments and waterways per cent. of total existing acreage as shown in L.C.C. Draft Development Plan, 1951. (This is the position before development and broadly indicates the open space existing for many years.)

Factors *b*, *c*, *d* were intended to measure social conditions and in addition it was considered that *c* would also measure the epidemiological influence of overcrowding apart from the economic conditions associated with poor housing conditions. The nutritional status of the boroughs is roughly indicated by *f*. Factors *g* and *h* were intended to measure the strength of the tuberculosis services. The occupational risk was intended to be indicated by *m*. Irish immigration could not be ignored and so factor *p* was introduced. Open spaces were referred to index *o*. Much of the information required for the calculation of the indices was only measured accurately at the Census of 1931, and it was for this reason that the analysis was based on the period 1931-33. Index *f* is based on 1938 because no other data were available, but it is not thought that at 1931 the borough pattern would have been different. The post-war position cannot be considered until the 1951 Census tabulations are available.

Essentially and briefly the statistical method of analysis is as follows:

If the movement of factor A is *not* independent of that of factor B, the two factors are said to be correlated. The strength of the correlation or association

(without prejudice to the question of whether or not the relationship is casual) is commonly measured by an index known as the coefficient of correlation (r). If the dependence is complete (*i.e.*, a change in A is always accompanied by a change in B), then r has its maximum value of unity. If there is complete independence, r is zero. Intermediate strengths of association are indicated by values of r intermediate between zero and 1. If A and B tend to increase together, r has a positive sign; if A increases when B decreases and *vice versa*, r has a negative sign. The coefficient is itself subject to sampling error and r must be significantly different from zero when this error is taken into account, for the association to be accepted.

In the calculations which follow consideration is first given to zero order correlation coefficients—*i.e.*, coefficients obtained by considering only the twenty-eight pairs of values of z and b, etc., without taking into account the possibility that both z and b may be related to c and that the first association may conceal the second. Later, *partial* coefficients are obtained for the association, for example, between z and b when the indirect effect of other factors is deliberately removed from the calculations—*i.e.*, effectively c, d, f, etc., are held constant while the variation of z with b is observed.

(I) *Mortality*

The zero order correlations were as follows:

Index z (Mortality from pulmonary tuberculosis) with:

b+0·725	c+0·688	d+0·679
	f—0·512	g+0·072
h—0·256	m+0·601	p—0·564
		o—0·296

It will be seen that there were statistically significant associations between mortality and all the social or economic factors.

Neither attendances at dispensaries nor the expenditure on these clinic services was significantly associated with mortality in 1931 (when treatment had less effect on mortality than now). This does not mean of course that the dispensary service was futile. It merely indicates that these factors were not sensitive indices of the influence of the service upon mortality.

The occupational risk gave strong association.

At first sight the proportion of Irish born in the population is of significance, but the correlation coefficient is unexpectedly negative. One might have expected more tuberculosis where there were more Irish born in view of known nationality (birthplace) factors, but on reflection it seems there is a simple and at least plausible explanation. Many of the Irish who come to work in London are engaged in personal service (often residential), so that relatively more would be found in better circumstanced boroughs (in 1931). On the other hand they are not numerous enough to raise the mortality rate significantly even if they do suffer a high tuberculosis incidence.

The inverse correlation of tuberculosis mortality with open space is *not* statistically significant.

None of the factors treated above is independent of the others, as will be

seen from the table of zero order correlation coefficients (Table V). Social class (*b*), for example, is strongly correlated with housing (*c*), nutrition (*f*), occupation (*m*) and Irish birthplace (*p*). If therefore there is an association between tuberculosis mortality and Irish birth there will also apparently be an association between tuberculosis and social class. Which is the direct and

TABLE V.—ZERO ORDER CORRELATION COEFFICIENTS

	b	*c*	*d*	*f*	*g*	*h*	*m*	*p*	*o*
z	+·725	+·688	+·679	−·512	+·072	−·256	+·601	−·564	−·296
b		+·719	+·836	−·591	+·183	+·070	+·626	−·464	−·135
c			+·903	−·636	+·261	−·193	+·915	−·403	−·463
d				−·583	+·349	−·007	+·816	−·469	−·352
f					−·209	+·313	−·470	+·402	+·333
g						+·075	+·210	+·114	−·174
h							−·201	+·133	+·174
m								−·333	−·378
p									+·345

The Position of the coefficient by row and column indicates the two indices between which association is measured.

which the indirect of the correlations demonstrated above? This question can be answered only by calculating the partial correlation coefficient between *z* and *b*, *z* and *c*, etc., while the influence of other factors is removed (the effect is as though, while *z* and *b* are considered, the other factors *c*, *d*, etc., are not allowed to vary among the boroughs and so cannot influence the direct association between *z* and *b*).

PARTIAL CORRELATION

Table VI gives values of the partial correlation coefficients found for the several multiple regression equations which were evolved.

In the analysis *g* and *h* were excluded owing to their lack of significance and *d* was omitted owing to its obvious overlap with *b* and *c*. The order of introduction of the factors was determined after experimentation with many combinations.

The second equation of Table VI takes into account the average social status of the borough and the average degree of mild crowding in dwellings—*i.e.*, the combined effects of economic level and facilities for exposure in the home. The small increment in variance explained by *c* indicates that much of the " housing " effect has been stolen by " social class " simply because it was introduced first and is highly correlated with housing. Fairly strong association with these two factors *together* is demonstrated with a contribution of 58 per cent. of sums of squares. In equation 3 the independent effect of nutrition (bodyweights of children) is introduced—*i.e.*, the effect not already

contained within social class and housing and " stolen " by those two factors—but no significant association exists and there is no further appreciable effect on the variance absorption. Equation 4 is a surprise, for the partial correlation coefficient for *m* is not significantly different from zero; on the other hand the consequent reduction in the partial coefficient for housing density suggests that the factor of employment in the specified occupations is not a direct tuberculosis risk so much as an association with the social background common to these low-grade occupations (*c* and *m* are highly correlated—see Table V).

Equation 5 shows that the introduction of the proportion Irish born produces a partial correlation coefficient of —·36, which is only just below the 5 per cent. significance level. The negative sign of the correlation coefficient and the fact that the coefficient of correlation with social class is reduced to ·39 indicates, however, that as suggested above we are measuring here not an

TABLE VI.—PARTIAL CORRELATION COEFFICIENTS BETWEEN TUBERCULOSIS MORTALITY 1931-33 AND FACTORS SPECIFIED

	b	*c*	*f*	*m*	*p*	*o*
Equation Multiple Regression	*Social class (proportions in IV. V)*	*Housing density (per cent. living more than 1½ per room)*	*Nutrition (mean weights per cent. London average)*	*Per cent. in high mortality occupations*	*Irish born proportion*	*Open space (per cent. of total borough area)*
1.	+·73	—	—	—	—	—
2.	+·45	+·35	—	—	—	—
3.	+·44	+·32	—·022	—	—	—
4.	+·44	+·20	—·0048	—·047	—	—
5.	+·39	+·20	+·046	—·043	—·36	—
6.	+·36	+·18	+·040	—·042	—·34	—·019

No.	*Regression Equation*	*Source of Variance*	*Sums of Squares*	*Degrees of Freedom*
1.	$z = ·0103b$	Variation explained by *b*	·2994	1
2.	$z = ·0067b + ·0044c$	Increment explained by addition of *c*	·0325	1
3.	$z = ·0067b + ·0043c - ·0020f$	*f*	·0003	1
4.	$z = ·0067b + ·0054c - ·00045f - ·0027m$	*m*	·0007	1
5.	$z = ·0055b + ·0050c + ·0041f - ·0023m - ·0036p$	*p*	·0306	1
6.	$z = ·0056b + ·0048c + ·0035f - ·0022m - ·0035p - ·00024(o)$	*o*	·0415	1
		Residual	·1653	21
			·5703	27

influence of place of birth so much as the influence of the social status of the borough. By the inclusion of *o* (Equation 6) the other coefficients for social class and housing, etc., are only slightly reduced, while the partial correlation coefficient for *o* is almost zero (compared with zero order —·296). This indicates also that *o* is not independent of *b* or *c*. As has been commented elsewhere (Benjamin and Nash, 1951), those who live in areas with little open space are commonly those who cannot afford to live in better residential areas and cannot afford good housing and other amenities.

The general conclusion to which we are led by these calculations is that there is very little gain in introducing the factors other than social class and housing density. Clearly we have not succeeded in finding any new factors related to mortality which are independent of economic status. None of the partial correlation coefficients is large enough to be regarded as statistically significant.

(II) *Morbidity*

Similar calculations were made for the notification rates 1931-33 in place of the mortality rates.

The zero-order correlation coefficients are:

Notification Rate a *with Variable*	
b	+·469
c	+·518
d	+·477
f	—·446
g	+·288
h	—·091
m	+·515
p	—·183
o	—·269

These resemble the coefficients found for mortality, but in general the associations are not so strong; especially the coefficients for *p* (Irish born) and *h* (expenditure on T.B. services) are much reduced, and the coefficient for *g* (clinic attendances per case) is increased. Inter-correlations of these variables have already been given in Table V.

The correlation coefficients for morbidity as distinct from mortality are shown in Table VII.

TABLE VII.—PARTIAL CORRELATION COEFFICIENTS BETWEEN PULMONARY TUBERCULOSIS NOTIFICATION RATE 1931-33 AND FACTORS SPECIFIED

Equation	*b*	*c*	*f*	*m*	*p*	*o*
1.	+·47	—	—	—	—	—
2.	+·16	+·29	—	—	—	—
3.	+·12	+·23	—·14	—	—	—
4.	+·11	—·06	—·21	+·20	—	—
5.	+·15	—·06	—·22	+·20	+·10	—
6.	+·20	—·11	—·22	+·22	+·14	—·15

No.	*Regression Equation*	*Source of Variance*	*Sums of Squares*	*Degrees of Freedom*
1.	$a = ·0091b$	Variation explained by *b*	·2337	1
2.	$a = ·0039b + ·0066c$	Increment explained by addition of *c*	·0724	1
3.	$a = ·0030b + ·0053c - ·023f$	*f*	·0152	1
4.	$a = ·0031b - ·0029c - ·034f + ·020m$	*m*	·0307	1
5.	$a = ·0037b - ·0027c - ·037f + ·020m + ·0018p$	*p*	·0077	1
6.	$a = ·0053b - ·0052c - ·035f + ·022m + ·0025p - ·0035(o)$	*o*	·0146	1
		Residual	·6901	21
			1·0644	27

There are some interesting contrasts with the picture presented by mortality in Table VI. In the first place the fit to the regression function is very much less close. None of the partial correlation coefficients is significant, though the correlation with social class is consistently little disturbed by the introduction of other factors. The coefficient for crowding is reduced from +·29 to —·06 when the factors of nutrition and employment in high tuberculosis mortality occupations are introduced. This does not necessarily mean that *f* and *m* are better indices of socio-economic status than *c*—it merely emphasises the non-independence of these indices so that *f* and *m* steal the

" crowding " effect. The " explained " variance increases by a negligible amount. The introduction of Irish birth does not affect the other correlation coefficients very much. The coefficient for p itself is surprisingly positive, but the difference from zero is not statistically significant and the unexpected positive sign is no more significant than the change of sign of the coefficient for c once we approach within the range of sampling fluctuation from zero. Social class alone makes a significant contribution to the variance.

Discussion

All evidence of the etiology of tuberculosis has pointed to an association with poor social conditions, but the elements in the environment involved and their relative importance have never been assessed with any degree of finality. This paper only adds to the pool of experience, not to any approach to finality.

Greenwood and Thompson (Collis and Greenwood, 1921) calculated partial correlation coefficients between certain social factors and tuberculosis mortality for 1911-13 in twenty-eight London boroughs. Their findings, in brief, were that conditions of factory employment and overcrowding in homes were factors influencing tuberculosis mortality.

The main findings of Hart and Wright (1939), who made a similar study, were that income and housing were significant and independent factors in the variation of tuberculosis mortality from area to area.

The present study, though ranging over a greater number of variables, hardly proceeds much farther. Tuberculosis is clearly a disease which is sensitive to changes in social factors and the multiple regression analysis suggests that the index or indices used to express socio-economic conditions might be chosen on a criterion of convenience alone rather than of other considerations from the available non-independent indices. On the whole, social class (employment in occupations in groups IV and V of the 1931 Census categories) and housing density appear to have the stronger claims. They give almost as good a prediction as any other combination and are readily available from census tabulations or from local sampling surveys which will tend to be carried out with increasing frequency. With regard to housing, Stein (1951) has found in Glasgow (since this study was made) that tuberculosis is more associated with persons *per dwelling* than with persons *per room*. The inference which might be drawn is that the important quantity is the *total persons exposed* in the household to which infection is introduced and not the degree to which they are crowded within the dwelling.

Summary

The differential incidence of and mortality from pulmonary tuberculosis in the London boroughs has been considered. Multiple regression analysis as at 1931 confirms earlier findings of the importance of socio-economic factors. It is not possible statistically to delineate satisfactorily the separate rôle of income, housing, nutrition, or occupational hazard, but it is found that consideration of the proportion in social classes IV and V (a measure of income) and housing density (more than 1½ persons per room) gives as good a guide as any to the expected variation of tuberculosis mortality between the

Boroughs at 1931. Morbidity variation is still largely unexplained. A brief account is given of the Borough trends since 1931 in relation to the social background.

I am grateful to Mr. C. A. Gould, B.Sc., M.C., for much help with tedious calculations; to Proffesor F. R. G. Heaf, who, while responsible for tuberculosis control in London, suggested this enquiry; and to colleagues, old and new, for helpful advice.

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"DENMARK'S SUCCESS AGAINST TUBERCULOSIS"*

By Niels Sjorslev

Medical Director of the National Association for the fight against Tuberculosis in Denmark

Even a short account of the fight against tuberculosis in Denmark must begin with a little history. The fight began as far back as 1875, when a sanatorium was opened at Refnes, on the coast of the island of Zealand, for treating children with scrofula. It was not until the middle of the nineties that the treatment of human tuberculosis began to take shape.

In the year 1900 it was possible to inaugurate the first sanatorium for consumptives in Denmark. This was the Vejle Sanatorium, with about 100 beds. Judged by the standard of those days it was a very modern affair. It was built for well-to-do patients able to afford the somewhat high charges required to run the institution. Less well-to-do people could not spare the money for sanatorium treatment, particularly unfortunate since it was among the poorer classes that tuberculosis spread most.

At a meeting on January 16, 1901, the National Society for Combating Tuberculosis was founded, with three objects:

(1) to prevent the spread of tuberculosis;
(2) to build sanatoria for the people; and
(3) to extend help to the families of those attacked.

At the same time the Government was induced to appoint a Commission to investigate the spread of tuberculosis and to recommend legislation to combat it. The mortality was shocking. In the towns, about 300 per 100,000 died of tuberculosis in all its forms, and about 200 per 100,000 of the deaths were from pulmonary tuberculosis alone. This was the average of all the towns in the country, large and small. In Copenhagen with its slums the death rate was much higher.

In the early 'thirties the demand for beds for tuberculous children had become insistent, and consequently the National Society built the sanatorium at Vordingborg with 118 beds, later increasing the number to 152. This is the sanatorium where British children are treated.

The terms of reference of the Commission, appointed in 1901 by the Government at the instance of the National Society, were to examine how the State, by means of legislation and suitable subsidies, could support the efforts being made to fight tuberculosis. The result of the work of that Commission was seen in two parliamentary Bills, one concerning measures against tuberculosis and one to provide State support for the building of tuberculosis institutions and for the treatment of patients. These Bills became law in 1905, and they were framed with such farsightedness that they are valid to this day, with relatively few amendments as the result of subsequent revisions.

Our laws on measures against tuberculosis seem somewhat scanty compared

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with the modern legislation of other countries. This is true particularly of the compulsory measures, but events have proved that even the few compulsory regulations which do exist have not as a whole been of much practical value. The explanation of this lies in the fact that the great majority of the Danish population is so well aware of the value of preventive measures against infection with tuberculosis that voluntary methods have achieved results that would hardly have been better if Denmark had had more elaborate compulsory legislation.

It is a fundamental principle of Danish law that the people have the right freely to dispose of their own person unless the contrary is expressly laid down by statute. Consequently, there is no obligation to submit to treatment for disease except when the law definitely states that such an obligation exists, for example, for certain categories of insanity and for persons suffering from infectious venereal disease.

Present legislation on tuberculosis is contained in two Acts of Parliament, of 1918 and 1952, which are concerned with measures for combating the disease and with State support for its treatment. Since the enactment of these laws a great deal of experience has been reaped in the fight against tuberculosis. This experience has made no change in the legislation, but has greatly affected its administration.

In Denmark people commonly have their own regular doctor. The number of physicians in Denmark, about one to every thousand of the population, combined with a well-established system of sick-clubs covering about 80 per cent., has enabled the whole population to have family doctors. If the patient has symptoms suggestive of tuberculosis, the doctor will always send him to a chest clinic for examination. If he is found to have tuberculosis, the chest clinic in consultation with the doctor will take steps towards instituting the necessary measures for his treatment, especially hospitalisation; in addition the chest clinic and the doctor will instruct the patient's family as to the measures advisable to prevent the risk of infection spreading. Through the medium of the chest clinic the doctor must always notify the local health officer of cases of tuberculosis of the lungs or larynx.

If a tuberculous patient fails to carry out the instructions given him by the chest clinic and the doctor, a report is made to the local health officer, who in turn notifies the local epidemic committee. This committee consists of the chief constable as chairman, the medical officer, and certain members elected by the local authorities; each borough has its own committee and each police district constitutes the area of an epidemic committee.

In the first place, the epidemic committee may decide as to the disinfection of rooms or other premises occupied by a tuberculosis patient and also of his clothing and bed linen. But in addition, where a tuberculous person lives or works under conditions of such a nature that they represent a distinct danger of infection to others, the committee may investigate and decide on measures to be taken. If the patient declines to comply with the decisions of the epidemic committee, the matter is laid before the Chief Epidemic Committee (consisting of the county governor, the county medical officer and three members elected by the municipalities). Even so, no measure may be ordered that will involve the separation of married couples against their will.

The law does not go into detail regarding the measures to be taken by the epidemic committee; but its wide terms (*e.g.*, that conditionally it authorises removal from the home) are assumed to provide authority for any limitation in personal freedom of movement necessary for the prevention of infection.

Compulsory hospitalization under our tuberculosis legislation cannot be put into effect through the police, nor can the police return to hospital a person who has left illegally. The only remedy for failure to comply with the decisions of the epidemic committee (including leaving a hospital when the epidemic committee has ordered the person to be admitted there) lies in penalties imposed by the ordinary courts. In such cases the courts may impose fines, confinement to prison up to two years, or imprisonment with hard labour not exceeding six months.

Should a person who has received an injunction of one kind or another deliberately endanger a spread of tuberculous infection the law makes him liable to imprisonment with hard labour not exceeding three years.

If the epidemic committee issues an order which causes loss to the person concerned by preventing him from doing his usual work or by involving him in a trading loss, he is allowed compensation based upon the principle of indemnity for financial loss.

We have other institutions in the fight against tuberculosis—first and foremost the chest clinics. Each county has its central clinic complete with physicians and nurses as well as the necessary clerical staff. Under it the central clinic has several sub-clinics, usually established at the local hospitals. All clinics are fully equipped with X-ray and other necessary facilities. The physicians at the central clinics hold consultations at the sub-clinics at regular intervals. The central clinics keep the main records. In Copenhagen and certain large provinical towns there are communal clinics each serving its own district. All the clinics work along practically identical lines.

The main object of the clinics is to find all cases of tuberculosis within their area and to diagnose them at the earliest possible moment.

The chest clinic is perhaps the most vital organ in the whole of our fighting system. A few of the clinics are associated with sanatoria and are attended by the physicians of those institutions. Most of them, however, are connected with a municipal hospital—a central hospital for an entire county—where there are specialists in the various branches of medicine.

The chest clinics have still another important object, B.C.G. vaccination, on which I should like to say a few words. I can give you a remarkable example from Denmark.

In a girls' college near Copenhagen there were 368 pupils, "teen-agers." One hundred and thirty of them were tuberculin positive as the result of spontaneous infection, 130 were positive after Calmette vaccination, whilst 105 were tuberculin-negative. Thus there were three groups of almost equal size. They were almost all taught by a female teacher who later proved to be tuberculous. This was during the war, and teaching went on in an hygienically bad, poorly ventilated basement, blacked out to serve as a bomb shelter in case of air-raids.

This is what happened to these girls: Of the 130 spontaneous positives, 105 were exposed to infection. Four contracted tuberculosis, with bacilli in

the lavage water; none died. Of the 105 negatives, 92 were exposed to infection. Seventy became inverters. Of these, 41 contracted tuberculosis, including 37 with bacilli in the gastric lavage water, and 36 had tuberculous processes on the X-ray films. Six developed regular consumption and one died. Of the 133 who were positive after the B.C.G. vaccination, 106 were exposed to infection. Two of them contracted pulmonary tuberculosis about a year after infection.

I can quote another example of the obvious value of this vaccination. From one particular county we used to receive 14 or 15 children every year with pulmonary tuberculosis. Now that the children in the county have been vaccinated we get none, or at most one a year.

In Denmark vaccination is voluntary, and yet about 99 per cent. of those found to be tuberculin negative in the mass examination have allowed themselves to be vaccinated.

In Denmark and the other Scandinavian countries, B.C.G. vaccination is widely practised. We vaccinate as many tuberculin-negative people as we can find, infants or old people. We believe that this vaccination provides relatively good protection from infection, at any rate for three or four years; if people then become negative again, we must revaccinate.

We consider it necessary to protect the population by means of vaccination. Gradually, as tuberculosis among human beings decreases, there are fewer opportunities for spontaneous infection, or, if you like, spontaneous vaccination. And now that bovine tuberculosis has been eradicated in Denmark, there is no longer the tuberculous milk that was another source of spontaneous vaccination.

In addition to the institutions I have mentioned, sanatoria, hospitals, chest clinics, nursing homes and so on, there remains a very important one—the State Serum Institute. This is a large establishment with laboratories for all the branches of bacteriology and aerology. But what interests us here in particular is the department for tuberculosis.

The Serum Institute tests all gastric lavage samples, all expectorates and all secretions that are sent in for testing for tubercle bacilli. The Serum Institute manufactures all the tuberculin used in Denmark and in many other countries as well. It makes the B.C.G. vaccine and supplies it to a number of countries, including Great Britain, I believe. I should like to point out, with the Lübeck disaster in mind, that the vaccine is prepared with every possible precaution. Only one man has the key to the B.C.G. laboratory—and perhaps there are some physicians here today who have visited it; they will know, then, that physicians will be admitted only after they have been X-rayed to make sure they are not suffering from T.B. All precautionary measures are taken to avoid infection of the cultures, nor have I ever heard of anything untoward happening.

A very considerable contribution to the fight against tuberculosis has been the eradication of bovine tuberculosis. It is somewhat outside the scope of this talk to mention this, but I would just say that measures against bovine tuberculosis were taken as long ago as 1888, and the campaign ended on June 1, 1952, when it was possible to declare the country's herds free of tuberculosis—not only clinically, but biologically, for all the animals are now

tuberculin-negative. For many years we have had strict regulations governing the sale of milk. In the towns, all milk had to be heat treated. The benefit of this is proved by the fact that no cases of glandular tuberculosis, so-called scrofulosis, have been recorded for several years; the disease has completely disappeared, and cases of meningitis have dropped to a fraction. This can be understood when one knows that the great majority of these diseases are caused by the bovine bacillus.

It is a very happy event for a country like Denmark, whose animal products are sold all over the world, to be able to guarantee that these products are free from tuberculosis. It has cost the farmers great efforts and large sums of money; and we may be quite sure that they reckoned the expense would pay in the end.

If in conclusion I try to explain why we have made so much progress in the fight against tuberculosis in Denmark, the question may naturally arise, is it because of the physicians and the work they have done? To that question I can give the unqualified answer: No! The medical contribution has been great, but without good tools and good working conditions the doctor will get nothing out of his efforts.

So we must say that without the understanding and readiness of the State to make the necessary grants we should not have done so much. There has been very profitable collaboration between voluntary organisations, as represented by the National Society, and the guardians of the State finances; parliament and government. Generally, the National Society conceived the ideas, and the State paid the cost of putting them into practice. We must remember that tuberculosis is a social disease. The better the social conditions, the more enlightened are the people, the better are the hygienic conditions. In Denmark, social conditions are good, and we have an enlightened population that adopts a positive attitude towards the fight against tuberculosis. In the years since the war there has been considerable housing shortage, according to our standards, but it is nothing as compared with conditions in many parts of the world. Slums, such as those in other large cities, are practically non-existent, and the great majority of the Danish working population live in bright and healthy dwellings.

All this costs money, of course. And if you wish to know how much it costs I can tell you that the annual expenditure on the fight against tuberculosis is 54,000,000 kroner, which is about the same number of shillings. This is equal to 13 kroner and 50 øre per head of the Danish population. However, I think it is money well spent; it will be repaid with interest, and it will save numbers of people from sickness and suffering.

LEONARDO DA VINCI AND THE BRONCHIAL CIRCULATION

By Leon Cudkowicz

From the Department of Medicine, Postgraduate School of Medicine, London

There has been a revival of interest in the bronchial circulation in the present century. Much that has been written, however, concerns the pure anatomy and the intra-pulmonary arrangement of the bronchial arteries (Miller, 1947). It was not until the patient observations by Daly and von Euler (1932), and Daly (1937), that the functional significance of this circulation was adequately appreciated, particularly in relation to the innervation of the bronchi and the pulmonary circulation as a whole. During the last few years some work has been done in an attempt to correlate the pathological changes in the lung parenchyma with lesions in the bronchial arteries, and a view is gradually emerging which endeavours to define ischæmic lung disease as an entity which might integrate certain lung conditions, such as emphysema, chronic bronchitis, and probably bronchiectasis, into the larger field of arterial disease and its resultant ischæmic states seen in other organs (Cudkowicz, 1951). In view of this conception it is perhaps remarkable that the bronchial arteries, which have been known for so many centuries, have been consistently ignored in lung pathology.

The History of the Bronchial Circulation

The classical monograph by Miller (1947) gives an admirable review of the history of the bronchial circulation. Miller points out that Galen (A.D. 130-201) first observed systemic blood vessels to the lung. In the " Tractat de arteria et vena dissectione " he wrote: " Arteria, quae secundum vertebras dorsi descendit, prima et tenuis propago, devitur ad thoracis partes, quibus pulmones adjacent, extremo vero abeunt in asperam."

Columbus (1559) disagreed with this contention, and stated categorically that the aorta does not send branches to the lung: " Ab aorta arteria ramus nullus, neque magnus, neque parvulus ad pulmones mittitur."

Dominico de Marchettis (1654) renewed interest in these vessels and discovered not only arterial but also venous radicles emerging from the lung and fusing with the systemic venous channels of the thorax.

In " Anatomia Patavii " he wrote: " Venas arterias possident pulmones, tum ob arteria venosa, tum a vena arteriosa, quae ut diximus per substantiam pulmonum dirimuntur sed observabiles sunt duae, aut tres arteriae ab arteria magna productae, quae substantium pulmonum propagantur."

Little more was heard of the bronchial circulation until Ruyisch, who claims for himself the primary honour of discovering the bronchial arteries. In his " Epistola anatomica " (1732) he denies the existence of bronchial veins.

The functional significance of these arteries appears to have remained unknown until 1808, when Reisseisen and von Sömmering defined the bronchial arteries as " die vasa nutritiva der Lungen."

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Thus a great deal has been written before the nineteenth century regarding the possible significance of these structures, but no references appear in these texts to the observations made on the bronchial circulation by Leonardo da Vinci nearly 500 years ago.

Leonardo da Vinci and the Bronchial Circulation

In the Quincentenary Exhibition held recently at the Royal Academy, London, and devoted to the drawings of Leonardo da Vinci, exhibit 301 shows a reproduction of a drawing from the Quaderni d'Anatomia II. Ir. (Royal Windsor Library) in which Leonardo depicts the trachea and the bronchial tree of the left lung with its accompanying bronchial arteries. The bronchial arteries are shown to arise from the aorta and to follow the individual bronchi to their termination (Fig. 1).

In the same drawing a circle is seen at the inferior margin of the lung which surrounds a terminal basal bronchus. A line runs from this circle to the adjacent script which, translated on p. 85 of the Catalogue, reads as follows: " Nature prevents the rupture of the ramifications of the trachea by thickening the substance of this trachea and making thereof a crust, like a nutshell, and it is cartilaginous and in the interior remains dust and watery humour." This, the catalogue emphasises, is one of the " earlier descriptions of a tuberculous cavity " (Fig. 3).

Closer scrutiny of this cavity reveals the additional and remarkable feature of small bronchial arteries within its periphery. It is impossible to say if Leonardo deliberately intended to show these systemic arteries reaching the walls of the cavity, or if the vessels depicted were merely branches destined to follow the bronchus containing the peripheral cavity. The latter explanation is probably the more likely.

Microscopy of the vasculature in the vicinity of tuberculous cavities has shown that the pulmonary arteries in the surrounding lung have usually undergone occlusive changes, and that the only true patent arteries in the walls of tuberculous cavities are dilated bronchial arteries (Wood and Miller, 1938; Wright, 1938; Cudkowicz, 1952) (Fig. 4).

The circle indicating tuberculous cavitation in this drawing is, therefore, of some interest in that it indicates, either by chance or deliberately, Leonardo's awareness of the existence of cavities, their relationship to the bronchial tree, and possibly also their correct blood supply.

Keele (1952) in his monograph on Leonardo's study of the heart and blood reproduces this same drawing and quotes from Leonardo's notebooks relevant passages appertaining to the bronchial circulation:

> " Why nature duplicated artery and vein in such an instrument, one above the other, finding themselves for the nourishment of one and the same member (the lung)."
>
> " You may say that the trachea and the lung have to be nourished, but if you had to do with a single large venarteria, this could not accompany the trachea without great interference with the movement which the trachea makes in dilatation and contraction as well as in length and thickness. Wherefore for this nature gave a vein and artery

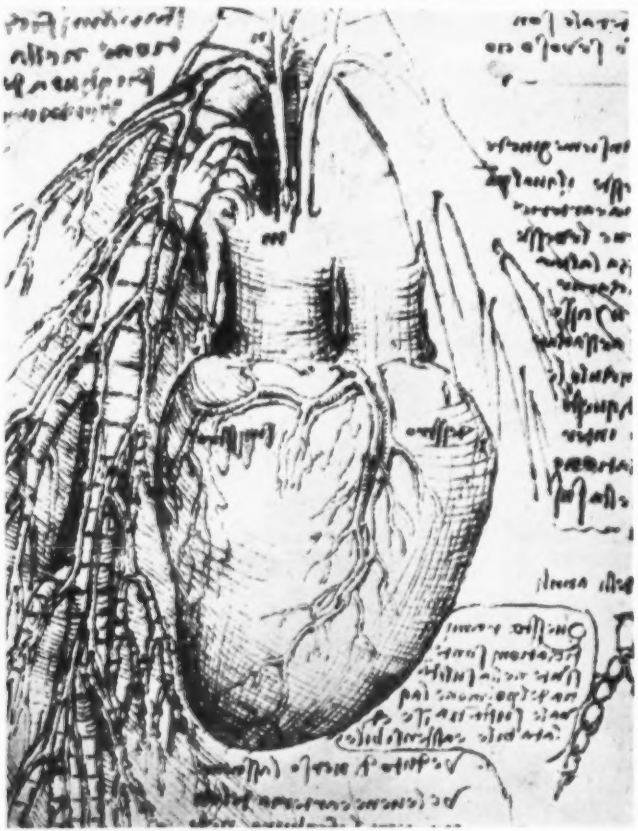

FIG. 1.—Drawing by Leonardo da Vinci of the heart, aorta and left bronchial circulation. The aortic origin of the left bronchial arteries is depicted as well as their course along the bronchial tree. (This is a mirror image drawing, therefore showing the apex of the heart and the aorta on the right hand side.)

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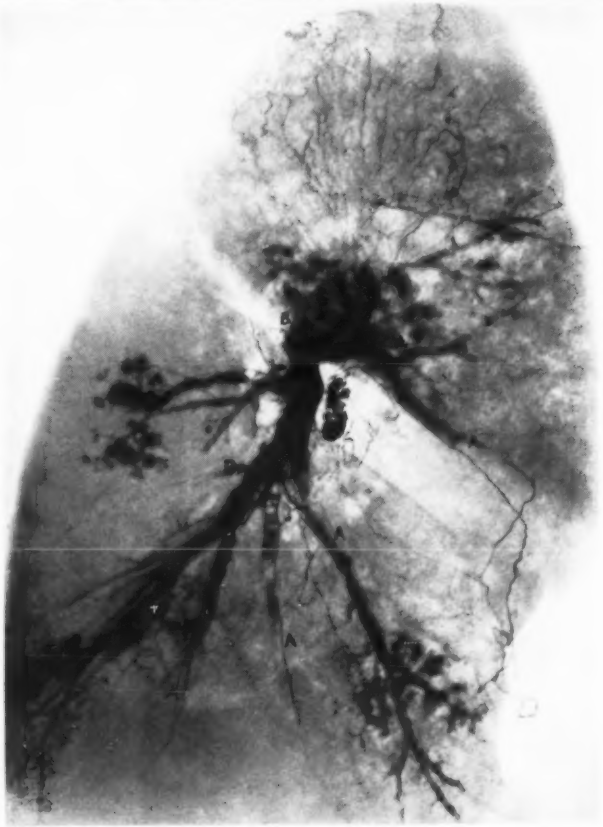

FIG. 2.—Lateral Radiograph of a left lung. The bronchial arteries and the bronchial tree have been injected with a contrast medium. (A) Bronchial arteries; (B) bronchial tree.

PLATE I

To face p. 24

FIG. 3.—Detail of drawing by Leonardo da Vinci of the bronchial arteries following the left basal bronchi. A tuberculous cavity is depicted in the centre of a left basal segment. (A) Bronchial arteries are shown in the vicinity of cavity.

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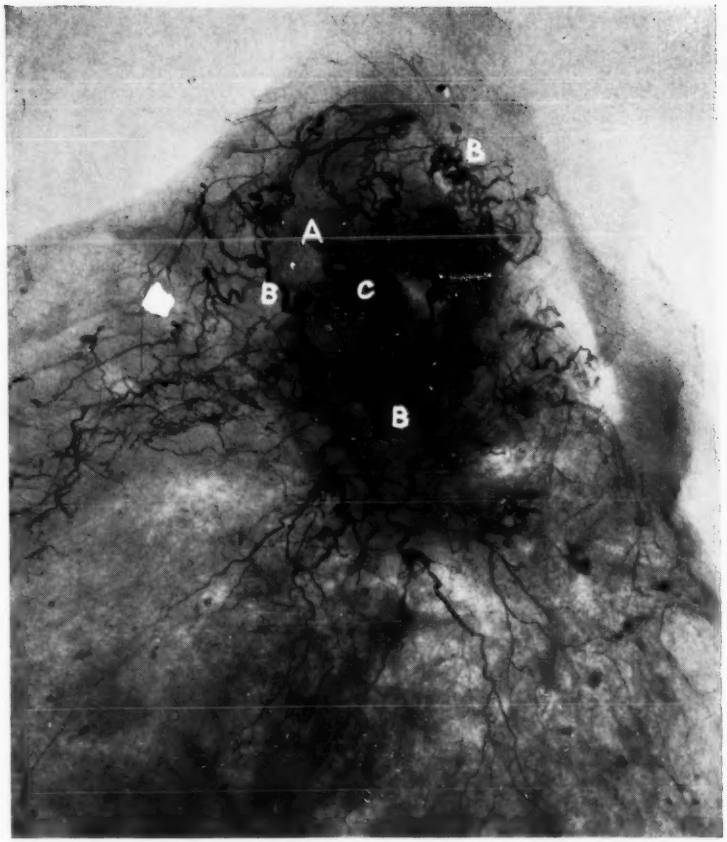

FIG. 4.—Lateral radiograph of a left upper lobe showing (A) tuberculous cavity; (B) bronchial arteries in considerable number in its vicinity; (C) a bronchial artery terminating in its wall.

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PLATE II

to the trachea which would be sufficient for its life and nourishment, and somewhat removed the other large branches from the trachea to nourish the substance of the lung with greater convenience."

(By trachea, Leonardo refers to the whole bronchial tree.)

This teleological view of the function of the bronchial circulation, expressed about 300 years before Reisseisen and von Sömmering's definition of the bronchial arteries as the vasa nutritiva of the lungs, is so modern in conception that very little can really be added. It bears testimony to his grasp of function of organs as much as the accuracy of his drawing emphasises his exceptional skill as an academic anatomist. Those who have tried to dissect the bronchial arteries, even under most modern conditions, are well aware of the hazards involved in tracing these delicate structures. Without the use of contrast mediums to outline the intra-pulmonary course of these vessels radiographically, and careful microscopy to show up the relationship of the bronchial arteries to the bronchial tree and other lung structures, modern studies of this circulation would be almost inconceivable (Fig. 2).

The drawing of the bronchial circulation by Leonardo, made at a time when such methods were unknown, is therefore quite beyond praise, and ought to have pointed the way to an earlier recognition of vascular lesions in relation to lung pathology. Yet Leonardo depicted the coronary arteries and emphasised their function in respect of the myocardium, but myocardial ischæmia as a concept in heart disease did not become known to medicine until the present century.

Leonardo da Vinci's mirror writing, his use of the "vulgar tongue," the intellectual indifference of his contemporaries, and the long incarceration in past centuries of his notebooks in private hands, may perhaps have been responsible for medicine's disregard of these remarkable observations. There are, of course, so many astounding facets to the life and work of Leonardo da Vinci which must overshadow his purely anatomical achievements. Those, however, who are concerned with diseases of the lung derive from this drawing of nearly 500 years ago inspiration and encouragement to elucidate the vascular factors in the genesis of lung disease.

I wish to express my gratitude to Mr. K. G. Moreman, A.R.P.S., for the reproduction of the radiographs; Sir Owen Morshead, the Librarian of the Royal Windsor Library; and the Secretary of the Royal Academy, London, for the photography of the original Leonardo da Vinci drawing.

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(B) bronchial arteries in considerable number in its vicinity; (C) a bronchial artery terminating in its wall.
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Depicted in the centre of a left basal segment. (A) Bronchial arteries are shown in the vicinity of cavity.
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INTRA-THORACIC AND INTRA-BRONCHIAL LIPOMATA

By Joseph Smart

From the London Chest Hospital

Lipomata have been recorded as occurring in almost every part of the body, most frequently in the subcutaneous tissue, where they cause no serious symptoms though they may be inconvenient and unsightly. Their occurrence within the chest, in the thoracic cage or intra-bronchially, is relatively rare, but here they may cause serious symptoms.

The object of this article is to record four cases of proved intra-thoracic lipoma and a further case, which in view of the history and similarity of the X-ray shadow was probably a lipoma, although it was never proved as the patient refused operation. There are two cases of intra-thoracic lipoma, one in the mediastinum and one within the pleura, and two other lipomata occurring in the bronchial tree, the latter being rarer than the intra-thoracic type. The earlier literature describes three types, those lying entirely within the thoracic cage; the dumbbell lipomata, with a portion of the tumour inside the thoracic cage and a portion extending through the intercostal space to the chest wall; and, thirdly, the mediastinal lipomata, which, if occurring in the upper part of the anterior mediastinum, may extend into the neck above the manubrium sterni. To these must now be added a fourth group—*i.e.*, lipomata occurring within the bronchial tree.

The first intra-thoracic lipoma was recorded in 1783 by Fothergill, and subsequently others have been reported, usually single cases (Abbot and Webb, 1935; Andrus, 1935; Andrus and Heuer, 1936; Auvray, 1918; Bariéty and Coury, 1950; Barrett and Barnard, 1945; Beatson, 1899; Bertoli, 1908; Beyers 1923; Bradford, Mahon and Grow, 1947; Büttner, 1941; Chiari, 1892; Clark, 1887; Conner, 1897; Cruveilhier, 1856; Czerny, 1875; Dissmann, 1950; Dor, Cristofari and Angelis, 1950; Ewing, 1928; Fitz, 1905; Fitzwilliams, 1913-14; Fontaine, Waitz, Buck, Riveaux and Mantz, 1951; Fulde, 1939; Garnier and Grosjean, 1903; Garrè, 1918; Graham and Wiese, 1928; Griffin and Guilfoil, 1948; Gussenbauer, 1892; Hall, 1949; Harms, 1920; Hess, 1926; Heuer, 1933; Jurine, 1815; Kittle, Boley and Schafer, 1950; Klemperer and Rabin, 1931; Lemon, 1925; Leopold, 1920; McCorkle, Koerth and Donaldson, 1940; Menghetti and Pasquali, 1951; Narr and Wells, 1933; Nylander and Kyllonen, 1950; Oestern, 1947; Plettner, 1889; Ruetz, 1932; Sauerbruch, 1925; Schanher and Hodge, 1949; Schinz and Gasser, 1933; Schreiber, 1880; Smart and Thompson, 1947; Soto, 1943; Swineford and Harkrader, 1942; Touroff and Seley, 1951; Vogt, 1876; Walker, 1936; Walzel, 1932; Watson and Urban, 1944; Wiper and Miller, 1944; Yates and Lyddane, 1930).

The earliest intra-bronchial lipoma appears to have been reported in 1854 by Rokitansky, who found, at autopsy, a sub-mucous bronchial lipoma

(*Received for publication October* 12, 1952.)

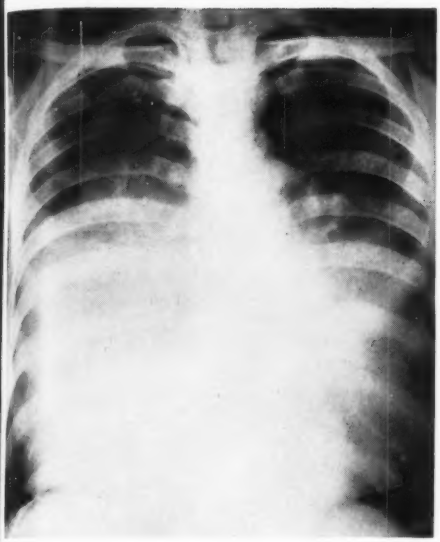

FIG. 1.—P.A. X-ray of chest showing mediastinal lipoma.

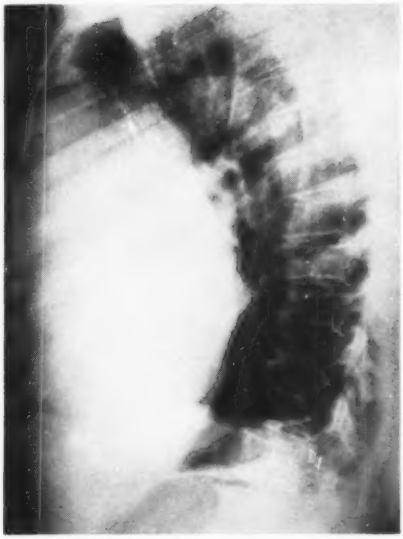

FIG. 2.—Lateral X-ray showing the mass anteriorly situated.

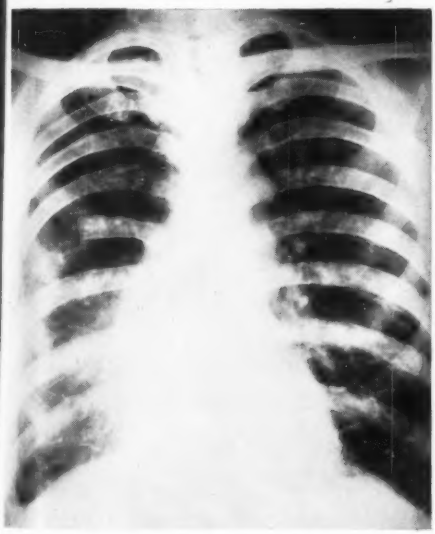

FIG. 3.—Post-operative X-ray after removal of tumour.

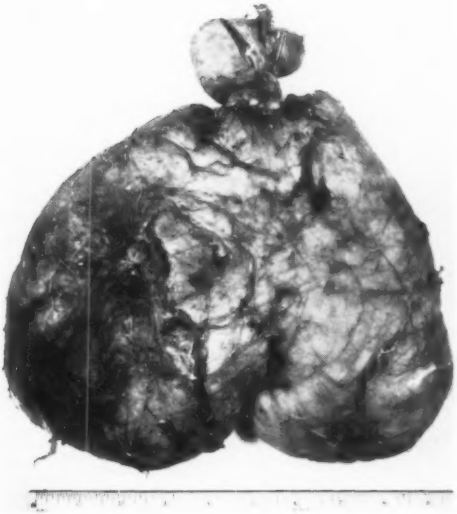

FIG. 4.—Photograph of lipoma.

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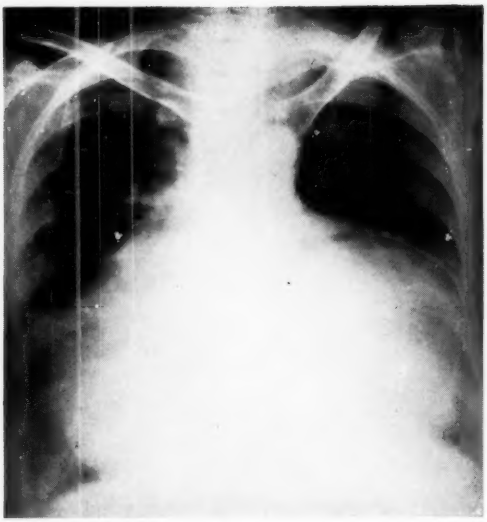

FIG. 5.—P.A. X-ray of chest showing large mass extending into both sides of the thoracic cage.

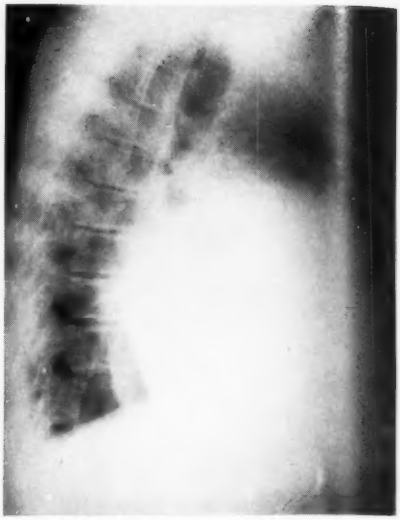

FIG. 6.—Lateral X-ray showing anterior situation of mass.

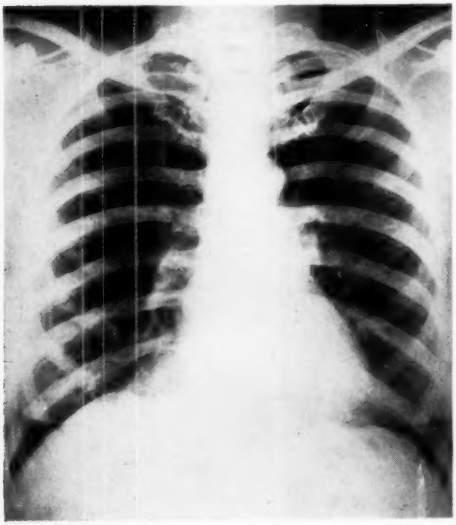

FIG. 7.—P.A. X-ray of chest showing tumour in right cardio-phrenic angle.

FIG. 8.—Tomogram of same case, demonstrating tumour at right cardio-phrenic angle more clearly.

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almost filling the lumen of the left main bronchus. In 1879, Labulbène mentions a lipoma of the bronchus containing spindle cells, but from then until 1922 no intra-bronchial lipomata appear to have been recorded. From 1927 onwards occasional cases have been reported (Carlisle, Leary and McDonald, 1951; Feller, 1922; Honig, 1934; Jackson and Jackson, 1932; Kernan, 1927; Lell, 1949; McGlade, 1939; Myerson, 1928; Terracol, Nichet and Guerrier, 1948; Touroff and Seley, 1951; Vinson and Pembleton, 1942; Watts, Clagett and McDonald, 1946; Wessler and Rabin, 1932; Whalen, 1947).

Case Reports

Case 1 (Previously reported in *Thorax*, 1947). F. H., a docker aged 52, came to the London Chest Hospital complaining of shortness of breath which he had noticed for two years and which was gradually increasing. For six weeks he had been unable to work. Previously he had always been healthy and had never had any severe illness. On clinical examination, his general condition was good, he had lost no weight and he was not breathless while lying in bed, but preferred three or four pillows. There was no cyanosis, no clubbing of the fingers, nor were there veins in the neck or abnormal physical signs apart from the chest. The physical signs in the chest were dullness in the anterior chest in the lower half, extending to the anterior axillary line on both sides, with absent breath sounds over this area and distant heart sounds. His blood pressure was 135/80 and the radial pulses were equal. X-rays, showed a large opacity in the chest extending to both sides, with fairly well-defined borders, through which lung tissue could be seen at the periphery of the shadow. The lateral X-ray showed it to be anteriorly situated. All further investigations were negative, including exploring the area with a wide-bore needle. The chest was explored and the lipoma removed in one piece. The total weight was 2,920 grammes, probably the largest lipoma removed in one piece so far. The patient has remained well subsequently. (Figs. 1, 2, 3 and 4.)

Case 2. M. E. W. This case is reported as a probable lipoma, although no proof was obtained histologically. The patient was an elderly lady who complained of increasing shortness of breath on exertion, but otherwise was well, with no loss of weight. On X-ray examination she was found to have an enormous tumour, with well-defined edges, in the anterior mediastinum. This was thought to be a lipoma in view of the similarity of the X-rays with those of the previously reported case, but the patient refused to consider operation and therefore the diagnosis remains unproven. This case is mentioned in view of the similarity of the X-ray shadow and the fact that this tumour was probably non-malignant, as the patient was well in herself with no loss of weight or appetite. (Figs. 5 and 6.)

Case 3. I. K., a woman aged 48, who had no symptoms but was found to have a shadow in the right cardio-phrenic angle on mass radiography. There was nothing abnormal in the past history. On examination no abnormal physical signs were found. The X-ray showed a shadow in the right cardio-phrenic angle, which was situated anteriorly, as shown by the lateral X-ray. A diagnostic pneumothorax on the right side showed the lung to be free from the tumour. Thoracotomy was performed, and the mass found to be a lipoma lying extra-pleurally against the pericardium. The capsule was opened and the tumour found to extend below the xiphisternum behind the right rectus sheath. This portion was removed as far as possible, the chest closed and the

rectus sheath opened. The remaining portion of the lipoma was then removed. It was found to be continuous with the extra-peritoneal fat. The post-operative course was uneventful. (Figs. 7 and 8.)

The following two cases are of lipomata occurring within the bronchial tree.

CASE 4. C. B., a man age 65, complaining of shortness of breath for ten years, which culminated in his collapsing when he was going upstairs. He was admitted to the West London Hospital and subsequently transferred to the London Chest Hospital. On examination, at that time, there was marked dyspnœa with white frothy sputum. The chest was resonant all over with added sounds to be heard throughout and a poor air entry. The cardio-vascular system was normal and the blood pressure 148/70. The sputum did not contain any tubercle bacilli or neoplastic cells. The hæmoglobin was 108 per cent. The X-ray showed emphysema with a large bulla at the right base and an opacity on the right side suggesting right middle lobe collapse. A diagnostic bronchoscopy was performed, and at the lower end of the trachea there was a pedunculated and lobulated tumour attached to the right lateral wall of the trachea. It was about half an inch in diameter and appeared to be covered by normal mucous membrane. It moved up and down on respiration. A biopsy was taken and this showed a metaplastic squamous epithelium covering the fibrous capsule of a mass of fatty tissue. Six days later a further bronchoscopy was performed and muco-pus aspirated from the right lower lobe in large quantities. The following day the tumour was removed under anæsthesia. The patient was induced with Pentothal and curare. A Magill tube was passed beyond the tumour and the cuff dilated in the trachea distal to the growth and the tumour removed piecemeal with biopsy forceps. The specimen showed one large and several small portions of tissue which could be aggregated to form a roughly spherical tumour 1.25 cm. in diameter. The surface showed lobular markings. The section showed the tumour to be covered by a metaplastic epithelium lying on a fibrous capsule. The mass of the tissue consisted mainly of fatty tissue of the adult type and some hypertrophied mucous glands. The diagnosis was one of intra-tracheal hamartoma.

Following operation, the patient recovered well and was discharged from Hospital ten days later.

The X-rays on admission showed an emphysematous condition of the right base with collapse of the middle lobe, and tomograms in the antero-posterior views showed a mass in the trachea which was also confirmed by the lateral tomograms. Following removal of the tumour, the X-rays cleared and the middle lobe also expanded. The patient has remained well since operation. A final bronchoscopy, six weeks later, showed no abnormality.

This tumour must be classified as a hamartoma, but was predominantly composed of fatty tissue and is therefore included in this series. (Figs. 9 and 10.)

CASE 5. A. N., a patient aged 60, was originally seen at the London Chest Hospital in November 1949 complaining of some indigestion with cough, and he was thought to have a diaphragmatic hernia. Investigations showed, however, that he had a paralysed posterior portion of the left diaphragm but no herniation through the diaphragm. He remained well apart from some bronchitis until November 1951, when he developed collapse of the left lower lobe, which then extended into the lingular of the upper lobe. This was thought to be caused by a pulmonary neoplasm. There was, however, no loss of weight

PLATE V

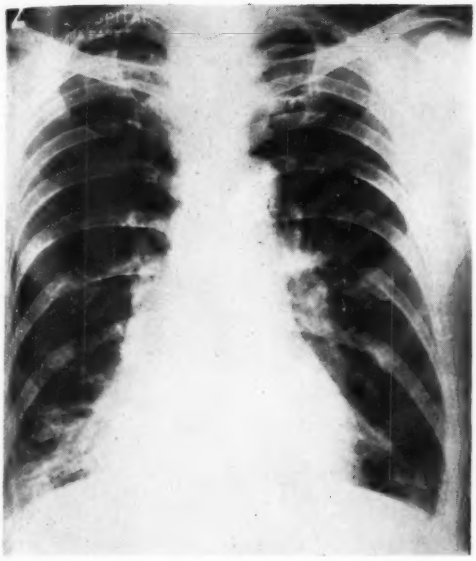

FIG. 9.—P.A. X-ray of chest showing infection at right base.

FIG. 10.—Lateral tomogram of same case showing pedunculated hamartoma in the trachea.

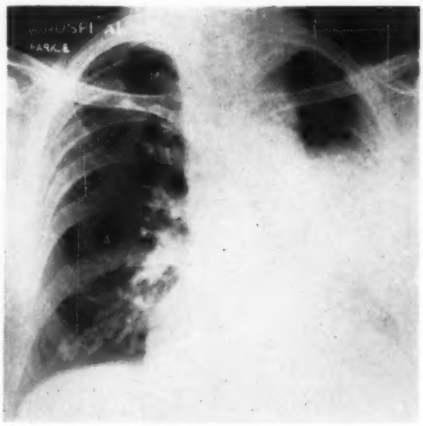

FIG. 11.—P.A. X-ray of chest showing collapse of left lower lobe and lingula.

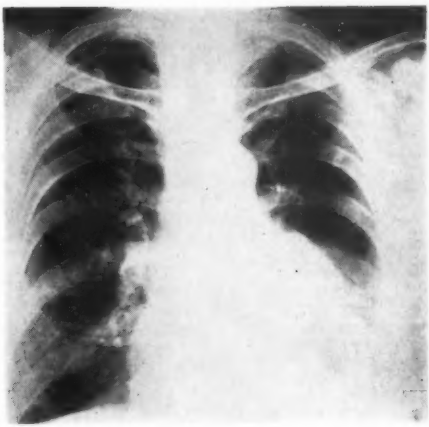

FIG. 12.—X-ray shows re-expansion of lingula, but left lower lobe remaining collapsed following removal of left intra-bronchial lipoma.

To face p. 28

or appetite, but he had marked cough and sputum with a raised white blood count and infection in the left lower lobe and lingular distal to the block. Bronchoscopy was performed and he was found to have a mobile, pedunculated tumour in the left main bronchus. Biopsy showed it to be a lipoma. A further bronchoscopy was later performed and the bulk of the tumour, including the pedunculated portion, removed piecemeal, but bleeding prevented the whole of the base being removed. Subsequently the cough disappeared entirely and the upper lobe re-expanded, but the lower lobe has remained collapsed. The patient feels and remains well and is averse to any further bronchoscopy in view of his fit condition. (Figs. 11 and 12.)

Discussion

Intra-thoracic lipomata are difficult to diagnose with certainty except by thoracotomy, unless they are of the dumbbell type coming through the chest wall, occurring in the upper part of the mediastinum presenting in the root of the neck, or growing within the bronchial tree; but, recently, symptomless lipomata have also been discovered by routine mass X-ray. In the past some of these tumours have been irradiated, but they are insensitive to X-rays and the correct treatment is removal of the tumour. The symptoms caused by the intra-thoracic and intra-bronchial lipomata are due to their size, but, owing to the smallness of the lumen of the bronchus compared with the size of the thorax, symptoms arise earlier in the intra-bronchial type. In this group, blocking of the bronchus frequently occurs, usually associated with collapse and infection of the lung tissue distal to the tumour which causes the presenting symptoms of cough, sputum and pyrexia. The intra-thoracic lipomata usually present with increasing shortness of breath owing to the size of the tumour.

Intra-thoracic Lipomata.—The initial symptom, as stated, in these cases is dyspnœa. The symptoms do not suggest malignancy because there is no loss of weight or appetite, though there may be pressure on the veins if the tumour is very large. Radiologically, lipomata may arise anywhere within the chest or from the mediastinum, and extend to both sides, and it may be extremely difficult to make an accurate diagnosis, but there are certain features which suggest a lipoma—*i.e.*, the tumour, which is often large, is frequently lobulated, it has clearly defined edges, and at the periphery lung markings can usually be seen through it. These tumours are, as a rule, outside the lung tissue, and a diagnostic pneumothorax will confirm this, as in case 3. The pathological investigations are normal, but if a lipoma is suspected and the tumour situated in a position which enables it to be explored with a wide-bored needle, it is at times possible to withdraw fatty tissue, which can be demonstrated by staining with Sudan III. The exact diagnosis is, however, usually in doubt until the chest is explored.

Intra-bronchial Lipomata.—In this group there is bronchial obstruction, with or without loss of weight, and the symptoms are similar to those caused by any benign tumour which gives rise to bronchial obstruction. The loss of weight, if it occurs, is due to toxæmia from suppuration distal to the block. The main symptoms are due to infection of the lung beyond the tumour, giving rise to cough, sputum and pyrexia, which may occur at intervals, but which usually persist when the tumour finally blocks the bronchus. Beyond the block the bronchi become dilated and infected. The white blood count is

frequently raised because of the inadequate draining of the infected area. The cough is paroxysmal and there may be very severe bouts of coughing, due to the fact that not only is the bronchus blocked but the tumour, being pedunculated, is free to move up and down the bronchus with each respiration. These lipomata can usually be removed bronchoscopically and they do not appear to recur quickly, but it may happen that the whole of the tumour is not removed by this means, and in the course of time it may recur, necessitating further bronchoscopy and removal, though up to date there has been no evidence of this. If, on the other hand, the lung is severely damaged distal to the block, lobectomy or segmental resection is the treatment of choice.

Carlisle, Leary and McDonald (1951) have reviewed the literature of the intra-bronchial type of lipoma, reporting a case. They also demonstrated that the normal bronchus contains fat external to the cartilage and to a lesser degree internal to it, and at times close to the groups of glands in the submucous layers. This fat occurs in the smaller bronchioles wherever cartilage is found, but not beyond. It is known that fatty tissue also occurs normally in the mediastinum and in the sub-pleural layers, so that lipomata occurring in these sites, as they do, probably arise from the fatty tissue normally present there. It is interesting to note that while lipomata in the subcutaneous tissue are usually multiple, occasionally being found in large numbers, intra-thoracic and intra-bronchial lipomata are usually single.

There are one or two other interesting points with regard to these cases. Intra-thoracic lipomata are much more common in men than in women and the age group is, as a rule, the carcinomatous age group. They usually occur within the major bronchi, more commonly on the left side than on the right, but Touroff and Seley (1951) reported a case of a lipoma which occurred within the lung and shelled out easily on operation, revealing a very small connection to the bronchus. Microscopically this lipoma was seen to be surrounded by bronchial tissue. From this it is clear that, while they commonly occur in the major bronchi, they may at times occur in the smaller bronchioles.

I would like to thank my colleagues Dr. E. H. Hudson for allowing me to report the case of intra-tracheal hamartoma and Dr. Lloyd Rusby the case of intra-thoracic lipoma. Mr. Vernon Thompson operated successfully on both these cases.

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RHEUMATIC TUBERCULOSIS

By Eli Davis

From the Rothschild Hadassah University Hospital, Jerusalem, Israel

The name "tuberculous rheumatism" (Poncet's disease) has been given to a condition of active lung tuberculosis in which rheumatic features are present. The rheumatism occurs generally at or near the onset of symptoms and signs of clinical tuberculosis. Sheldon in 1946 and Seligson in 1945 gave good accounts of the disease with illustrative cases. The five cases described below show the range of manifestations encountered, and emphasise the important inferences to be drawn—namely (*a*) that in atypical acute rheumatic fever and subacute rheumatism the possibility of tuberculosis should be considered, and (*b*) that the diagnosis of rheumatic fever does not exclude the possibility of co-existing tuberculosis (and *vice versa*).

Cases

The salient features only of the cases are given.

Case 1. A boy aged 6, with a strong family history of rheumatic fever, began to be ill with a dry cough. One month later he suffered from a sore throat and complained of fleeting pains (without swellings) in the right ankle, knee and hip. The erythrocyte-sedimentation rate (E.S.R.) (Westergren) was 20 mm. in one hour. He was admitted to hospital for sub-acute rheumatism, but fever persisted despite salicylates. He had a positive Mantoux test (1 : 10,000), and on radiography a large shadow was seen around the right hilum. Observation confirmed the diagnosis of active tuberculosis.

Case 2. A girl aged 15 was hospitalised for acute pericarditis going on to effusion, complicated by collapse of the lower lobe of the left lung. She developed pain in the left shoulder (without swelling) for one night only. The condition was treated as acute rheumatic fever. Fever subsided and the abnormal signs disappeared with the exception of tachycardia, which persisted. Five months after admission, râles were heard at the apex of the left lung and tubercle bacilli were recovered from her gastric juice.

Case 3. A girl aged 19 had had pains in her hands and feet for two years, and, for several months, swellings of the joints of the hands, knees and ankles. She had more movement and less pain than would have been expected in rheumatoid arthritis. On examination, râles were heard at the left apex, and skiagrams showed infiltration, probably recent, which was progressive. E.S.R. was 80 mm. in one hour. Yet the usual symptoms of lung tuberculosis were absent.

Case 4. A boy of 18 was treated in hospital for three months with acute rheumatic fever with a characteristic history and typical signs. Two months after discharge cough began and four months later widespread phthisis was present.

Case 5. A man aged 39 gave a six months' history of pain in the right lumbar region. For the past two months he had had persistent low-grade

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fever and weakness, and also joint pains in the hands and feet but without any redness or swelling. For the past six weeks he had suffered in addition from pains "in his muscles." The pain, however, was less on movement of his joints, and increased when he rested. He was admitted to hospital with the diagnosis of rheumatic fever. Examination revealed low-grade fever, but abnormal physical signs were absent. The first mitral sound was accentuated, but there were no murmurs. Blood pressure was normal. E.C.G. showed biphasic S.T. complex in lead III. Radiography showed infiltration in the upper zone of the left lung and enlargement of the left ventricle. Joint pains persisted, but were not influenced by aspirin. E.S.R. was high. A blood count showed polymorphic leucocytosis. A provisional diagnosis of virus pneumonia was made. However, tubercle bacilli were found in the gastric juice. Follow-up proved progressive phthisis. Two months after admission his weight began to increase and his sputum became (and remained) negative. E.S.R. kept high, however. Joint and muscle pains disappeared almost completely.

This case of active phthisis was regarded for eight weeks as rheumatic fever. It is not possible, however, to exclude the diagnosis of rheumatic fever despite the onset at age 39, because there were present persistent joint pains and fever (although uninfluenced by aspirin), the left ventricle was enlarged and E.S.R. remained high even when the phthisis was improving.

Discussion

The above cases show well the varieties of rheumatic features encountered. Cases 1 to 4 were seen among 750 personal cases of pulmonary tuberculosis in London, England, while case 5 was referred to me by Dr. P. Weyl and Dr. I. Rakover of the Hadassah Tuberculosis Hospital, Jerusalem. Thus in case 1 the cough was at first regarded as due to the upper respiratory infection which so often is associated with acute rheumatic fever. Case 2 is of interest in that Sheldon stated that while a fleeting pericarditis without an accompanying arthritis may indicate recent tuberculous infection, he had not seen any such instances recorded. In my cases (as in other series), as the disease progressed certain atypical features threw doubt on the diagnosis of acute rheumatic fever—thus in cases 1 and 5 there was no response to salicylates, in case 2 at most only one joint was involved, while in case 5 movement eased the joint pains.

I suggest that it would be more correct to call these cases of Poncet's disease rheumatic tuberculosis rather than tuberculous rheumatism, because tuberculosis is the major disease concerned. It must be pointed out, however, that Poncet and Leriche called their book *Le Rhumatisme Tuberculeux*.

In 1947 I reported that, in a personal series of 725 patients with active pulmonary tuberculosis and tubercle bacilli in the sputum, 27 patients gave good evidence of having suffered from rheumatic fever. Of the 27 cases 6 were proven to have mitral stenosis. It is possible that other cases of mitral stenosis existed in this group, but proof was lacking. During the period when these 725 patients with tuberculosis were seen, I saw 583 other patients with convincing histories or signs of rheumatic fever or chorea. Of these 583, 3 developed lymphocytic pleural effusions and 3 others stated that they had been treated for active pulmonary tuberculosis from which they recovered.

Summary

Five cases of rheumatic tuberculosis (Poncet's disease) are described showing the various clinical features encountered in this condition. These cases demonstrate that (*a*) in atypical acute rheumatic fever the possibility of tuberculosis should be considered, and (*b*) the diagnosis of rheumatic fever does not exclude the possibility of co-existing tuberculosis (and *vice versa*).

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BRONCHIECTASIS CAUSED BY MUSTARD GAS

By N. Wynn-Williams

From the Chest Clinic and General Hospital, Bedford

The main accounts of bronchiectasis published since the first World War scarcely mention mustard gas, although a number of papers have established its reality as an etiological factor and many cases of bronchiectasis due to this cause have been reported by themselves. This article sets out to show that in a general series of patients suffering from this disease, and coming under observation at the present time, mustard gas must be judged of some importance.

The Literature

Mustard gas (dichlorethylsulphide) was synthesised in 1886 by Meyer, who recognised its toxicity for the skin, eyes and respiratory tract. Lynch, Smith and Marshall (1918) confirmed its irritant properties and also observed that absorption into the blood stream through the skin or respiratory tract could occur with marked systemic effects. Warthin and Weller (1919) found that 95 per cent. of men who had been gassed showed involvement of the respiratory tract. Laryngitis and bronchitis arose, and in more severe cases congestion and œdema of the lung progressed to localised atelectasis due to bronchial plugging. Secondary infection caused purulent bronchitis and bronchopneumonia. Loeper (1919) proved at autopsy that pulmonary sclerosis and bronchiectasis might follow the inhalation of mustard gas.

Probably the first bronchographic proof appeared in the French literature, and Bonnamour, Badolle and Gaillard (1929) collected a number of cases that had been confirmed by this means in their monograph on the pulmonary uses of lipiodol. Furthermore, Carr, Denman and Skinner (1947) described the results of an accident during the second World War in which a number of people were exposed to such fumes. Bronchograms were done on 144 patients, 46 of whom showed considerable or extensive bronchiectasis.

Among the better-known descriptions of bronchiectasis Farrell (1936) reported one case; Fletcher (1935) two cases; Perry and King (1940) one case; Bradshaw, Putney and Clerf (1941) two cases; and Kinney (1947) one case. No other author was found who mentioned gassing as an etiological factor in a general review of this disease. The total of seven patients forms less than 1 per cent. of the series in which they occur.

Material on which the Present Study is based

This study is based upon 166 cases of bronchiectasis confirmed by bronchograms. The series included 29 children under the age of 15 and 137 adults, of whom 73 were men and 64 women. These patients were referred from a population of about 150,000, and represented all who came under observation with this diagnosis during the five-year period from 1947 to 1951. Eight men

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with bronchiectasis almost certainly caused by mustard gas were found. Six of them had been admitted to hospital after gassing with injury to the eyes and skin; two were treated for burns but did not require admission.

Eight other men were seen with histories and physical signs suggestive of a similar diagnosis, but they could not be included for the following reasons. Five had ordinary radiographic evidence of bronchiectasis, but three of them were unconfirmed by bronchogram and two, previous to gassing, had had pneumonia. The remaining three had normal radiographs and iodised oil failed to demonstrate bronchial dilation.

Clinical Findings

The two youngest patients were aged 51 and the oldest 63. None of them had had any severe respiratory illness or symptoms referable to the chest before being exposed to mustard gas in 1917 or 1918. Four had suffered from constant cough and sputum since this time, four had had intermittent cough and sputum mainly affecting them during the winter months. When first seen six had had constant cough and sputum for at least ten years. All had been diagnosed as having chronic bronchitis, but only one had been radiographed previously. The smallest regular volume of sputum while under observation was half an ounce, the largest 2 ounces. Intercurrent respiratory infections at least doubled this quantity. A mixed bacterial flora was found in all instances, but no tubercle bacilli. The sputum was fœtid in one patient when first seen, but regular "tipping" removed the fœtor and also reduced the average daily volume. Blood staining of the sputum was noted in four, but only one had suffered from a frank hæmoptysis of 8 ounces.

Abnormal physical signs were present in all these patients, the most obvious of which was emphysema. Hyper-resonance (particularly of the bases) helped to mask the common localising signs of bronchiectasis. Persistent râles were, however, present over the bronchiectatic area in seven patients. Clubbing of the fingers was observed to be well developed in four and slight in two. The vital capacity was taken in all; the lowest was 1,300 c.c. and the highest 3,600 c.c., only one other being above 3,000 c.c. Dyspnœa was the most prominent symptom and was present in four on moderate exertion, in two on slight exertion, and one was unable to work for this reason. The degree of dyspnœa was out of proportion to the extent of the bronchiectasis and appeared to be associated with generalised rather than compensatory emphysema.

The radiographic appearances were abnormal in all. Local or general emphysema was present in six with markedly flattened diaphragms in three. Changes otherwise were typically rather slight and consisted of localised broadening and crowding of the pulmonary markings in six, with ring shadows of varying sizes in three, and nodulation up to 1 cm. in diameter in four. Larger and more irregular opacities were present in two, but there was no instance of the triangular shadows cast by segmental or lobar collapse apart from the right cardiophrenic angle being obscured in two.

Except for one, these patients were still in employment, but two were carrying on light jobs only with great difficulty. The period under observation

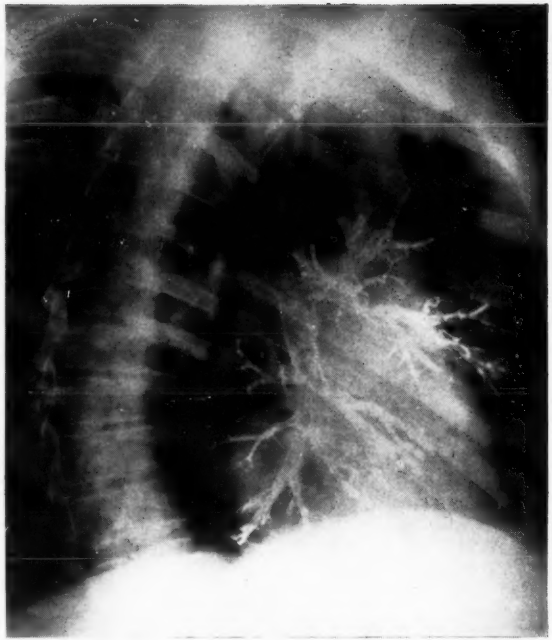

FIG. 1.—Right posterior oblique bronchogram showing bronchiectasis of the right middle lobe and absence of normal tapering of the basal branches of the right lower lobe.

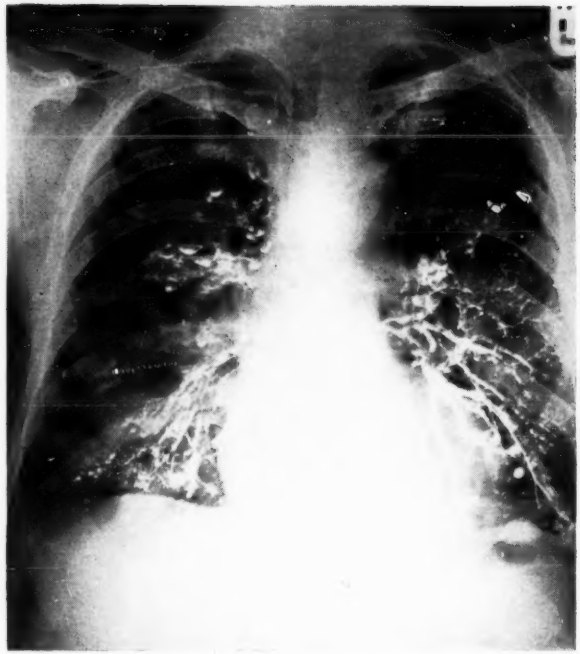

FIG. 2.—Antero-posterior bronchogram showing saccular dilatation of the right upper lobe and mainly cylindrical dilatation of the right lower lobe. Cylindrical and saccular dilatations are also present in the left lower lobe.

averaged three and a quarter years, during which time six lost considerable periods from work during the winter months because of bronchitis, and pneumonia occurred in one patient. Sinusitis was present in two, but none had asthma. Six had suffered from pneumonia at some period subsequent to their gassing.

The diagnosis was confirmed in each by bronchograms, but in three only one side was outlined owing to the patient's small vital capacity. Bronchiectasis was present in nineteen lobes, and among the five with bilateral bronchograms it was right-sided in one, left-sided in one and bilateral in three. The type of ectasis was cylindrical or fusiform in four, and a varying degree of sacculation was present in the remainder. Fig. 1 shows an example of the former type and Fig. 2 of the latter.

The prognosis of these patients as judged by dyspnœa on exertion would appear to be bad. While under observation two have become more breathless, though no change has occurred in the others. It seems likely that cor pulmonale will be the cause of death in a considerable proportion of these men. The response to treatment by postural drainage and antibiotics was similar to that of patients with bronchiectasis of other etiology.

Discussion

It is seen that bronchiectasis caused by mustard gas was found in 5 per cent. of a general series of 166 patients coming forward with this disease during a recent five-year period; it was the relevant etiological factor in eight out of seventy-three men. There can be little doubt that mustard gas was the cause of the bronchiectasis in these cases, as their histories were quite definite. Apart from these patients it was noted that a number of men mentioned gassing as one factor in their history, but this was rendered irrelevant by the occurrence of other severe respiratory illness. There was also a class who suffered from " winter " bronchitis without bronchiectasis, starting from the time of gassing and not previously. It is not clear why so little attention has been paid to this etiological factor in the past. Possible reasons are that, as in this series, such patients with few exceptions have not been radiographed and have been accepted as suffering from bronchitis; that only during the last decade have their symptoms become sufficiently disabling to demand investigation; and that, obviously unsuitable for surgery, few have been referred to the large hospitals from which most papers on bronchiectasis have been compiled.

Bonnamour *et al.* (1929) held that bronchiectasis caused by mustard gas was always cylindrical, but the dilatations found in this investigation were of varied types as reported by Carr *et al.* (1947). The only difference in the clinical findings between bronchiectasis due to mustard gas and to other factors lay in the degree of dyspnœa on exertion, which was disproportionate to the extent of dilatation. Its source was emphysema and in the winter superadded bronchitis. It is likely that mustard gas, apart from causing bronchiectasis, renders the bronchi more susceptible to respiratory infection with the subsequent development of emphysema. The recognition of mustard gas as a factor causing bronchiectasis may be of some importance to the patient, as occasionally it is possible to persuade the Ministry of Pensions to admit attributability. Such an appeal was made by five of these patients, with success in two.

Exposure to chlorine, though capable of causing broncho-pneumonia (Rathery and Michel, 1915; Sergent and Agnel, 1915), was not found to be an etiological factor in any patient. However, only two patients were seen, both with bronchitis and emphysema, who mentioned chlorine in 1915 as a possible factor. Both had experienced an interval of good health after recovering from gassing before their symptoms started.

Summary

Among a general series of 166 patients with bronchiectasis eight were found who traced the start of their symptoms to exposure to mustard gas.

Apart from dyspnœa on exertion due to emphysema the clinical and radiological findings were similar to those found among patients with bronchiectasis following other etiological factors.

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CARDIOSPASM: INTERMITTENT: AN INITIAL MANIFESTATION OF CARCINOMA OF THE CARDIA

By N. Asherson

From the Royal National Throat, Nose and Ear Hospital, and the Institute of Otology and Laryngology

This prodromal syndrome receives scant reference in the literature.

Intermittent or transient cardiospasm may be an initial manifestation of carcinoma ventriculi: such paroxysms may precede gastric symptoms for a considerable period (in this case, for over two years) and may even lead to an exploratory thoraco-abdominal laparotomy before the real nature of the disease is disclosed. In the unusual case to be described, this was the precise sequence of events.

For a period of about two years, the patient complained of vague discomfort on swallowing referable to the pharynx and to the upper end of the œsophagus. Radiography and œsophagoscopy revealed a normal pharynx and œsophagus. Subsequent X-ray examination revealed a dilated œsophagus, and the diagnosis of cardiospasm was made. Under screening the dilatation was observed to be of the spasmodic and peristaltic type; the X-ray repeated at a later date showed no dilatation of the œsophagus.

The cardiospasm occurred intermittently (*i.e.*, was transient). It could always be precipitated by the patient. Whenever he ate a piece of apple symptoms developed and, on screening, the œsophagus was observed to be extensively dilated—a typical cardiospasm. Over two and a half years these findings were constant—periods of normality of the œsophagus interleaved with periods of abnormality, typical of the cardiospasm.

Œsophagoscopy (third in two and a half years) revealed a normal œsophagus. In view of his persistent symptoms, it was decided to perform a Heller's operation. Laparotomy revealed extensive carcinoma of the cardia with secondary glands in the lesser omentum. The spasmodic transient cardiospasm was caused by reflex nerve irritation due to the large growth in the stomach. There were no gastric symptoms: he had no anæmia. All he complained of was difficulty in swallowing certain solids—*e.g.*, an apple.

Case Record

Male, 44, seen first in August 1951, with a two and a half years history of difficulty in getting down bread or anything dry. This is intermittent. He can feel an obstruction referred to the upper part of his throat, but the food goes down. There is no pain on swallowing: no regurgitation of food: no loss of weight: no alteration in voice: no cough or hæmoptysis: appetite is diminished: some nausea but not sick: bowels not regular.

Eighteen and six months ago X-ray of the œsophagus (Fig. 1, 1A) and œsophagoscopy proved negative. The symptoms persisted.

The X-ray report read: " There was no delay through the œsophagus to either thin or thick barium except that at the lower part some slight achalasia

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was seen with very slight dilatation of the lower end of the œsophagus. The outline, however, appears to be quite smooth and there was no actual obstruction to the passage of barium into the stomach, the upper part of which was outlined and in the supine position shows a normal outline.

"Even in the supine position barium passes freely and I could find no evidence of an organic lesion."

He had no skin lesion (scleroderma).

The second X-ray report stated: "Opaque swallow showed normal œsophagus, but when he swallows a piece of apple or bread, the œsophagus under the screen is observed suddenly to dilate, giving the appearance of a cardiospasm." (Figs. 2 and 3.)

Now, œsophagoscopy (the third) revealed a normal, non-dilated œsophagus with the cardia tightly constricted; a size 23 bougie was passed with difficulty and when withdrawn no blood was observed. Subsequent dilatation by a Hurst's bougie daily led to no improvement.

Further radiological report was as follows:

(1) Thin barium: there was no abnormality in the passage of thin barium. There was no hold-up at the cardia or the crico-pharyngeus. Films taken.

(2) Dry bread swallowed and barium given. No sticking produced, normal passage.

(3) After eating apples: has to eat more than one to produce sticking sensation.

(4) Barium swallow: there is a definite cardiospasm, and this is illustrated in the films. (Fig. 4a and b) "taken in Trendelenburg position."

Conclusion: This case is, therefore, a cardiospasm, which for some reason is produced by dry foods and apples. The peristalsis in the œsophagus is marked, and when the patient says that the food is passing this can be seen on the screen.

February, 1952

It was proposed to perform a Heller's operation, but laparotomy revealed that the cardiac part of the stomach was occupied by a large, hard mass; there were palpable glands along the lesser curvature and nodules in the pelvic floor. The liver was free from palpable secondaries.

Comment

The intermittent cardiospasm was for two and a half years the only sign of a carcinoma of the cardiac end of the stomach. The only symptom the patient complained of was "sticking of the food in the upper part of the neck," especially a piece of apple or any dry morsel of bread.

I am indebted to Dr. T. V. Crichlow for the radiographic opinion and the X-ray plates.

Sir Heneage Ogilvie (1947), drawing attention to the relation of cardiospasm to carcinoma of the cardia, writes: "A diagnosis of cardiospasm should never be made in an adult with a short history until the possibility of a high gastric cancer has been excluded."

While this paper was in the press, W. D. Park, (1952), reported a case with a similar history in a series of seven cases of carcinoma of the cardiac part of the stomach. It occurred in a female aged 34. The diagnosis was at first cardiospasm, as a result of an opaque swallow, which was repeated at a later date. Four months later, laparotomy revealed the causal cardiac carcinoma.

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FIG. 1.—Œsophogogram. No dilation. 7.5.51.

FIG. 1A.—X-ray of Pharynx. No abnormality.

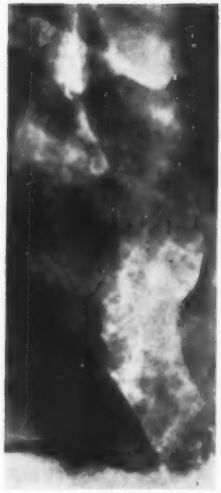

FIG. 2.

FIG. 2.—Intermittent Cardiospasm due to Carcinoma of the Cardia. A double exposure, superimposed showing peristalsis and movement of the œsophagus on swallowing a piece of apple. 3.9.51.

FIG. 3.—Cardiospasm: two separate exposures. (On other occasions, the œsophagus showed *no* dilation.) 13.9.51.

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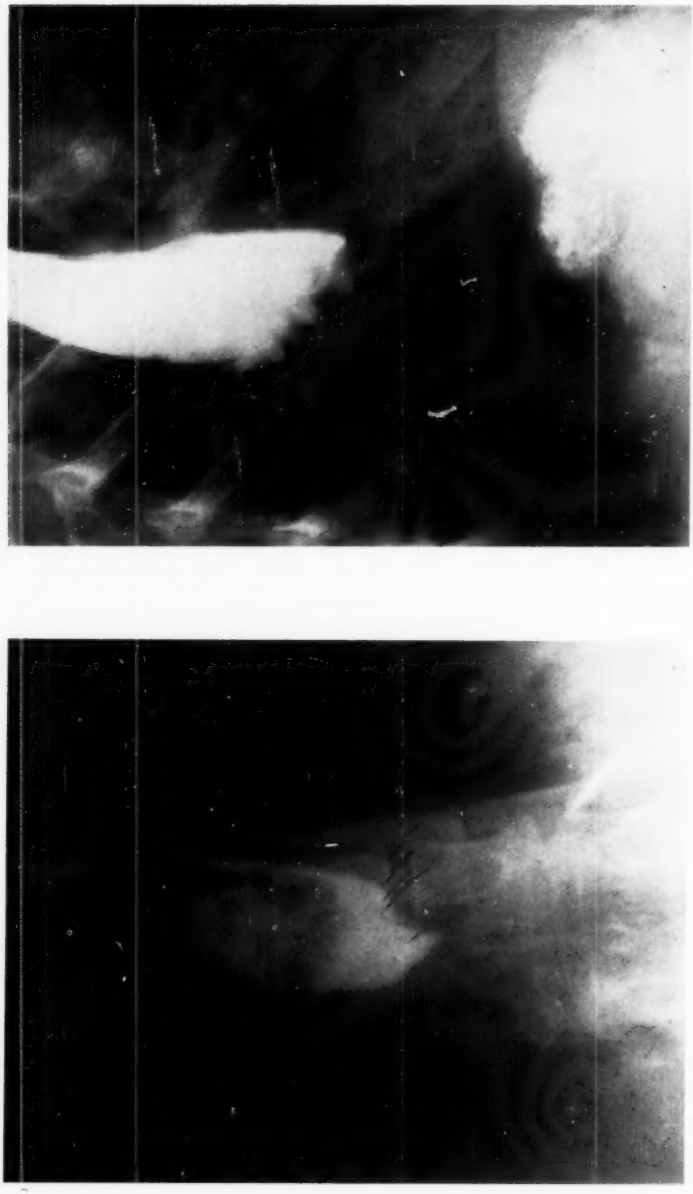

FIG. 4 AND 4A.—Œsophogograms, at a later date. (Taken in the Trendelenburg position.)

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SERIAL HÆMAGGLUTINATION TESTS DURING THE TREATMENT OF PULMONARY TUBERCULOSIS

By D. A. L. Bowen and R. C. Jennings

From the Department of Pathology, London Chest Hospital

In 1948 Middlebrook and Dubos described a hæmagglutination reaction for the detection of antibodies against the mammalian tubercle bacillus.

Hinson, Richardson Jones and Chamberlin (1952), writing in this journal, reviewed the literature and recorded their findings in 130 unselected hospital cases of pulmonary tuberculosis, using a modification of the originally described hæmagglutination reaction. Briefly, their results showed that the proportion of positive reactions increased with the anatomical extent of the disease, but that they were uninfluenced by the age of the patient and that the test in its present form had no value in the diagnosis or prognosis of pulmonary tuberculosis. Similar results were obtained by Fleming, Runyon and Cummings (1951).

Most writers on the subject are agreed that the finding of positive reactions in non-tuberculous controls and negative reactions in some tuberculous cases, although few in number, invalidate the test as a diagnostic aid. However, it is of value in the differential diagnosis of obscure radiological shadows in the chest. Previous investigators have recorded single readings of the hæmagglutination titre from various groups of patients. The value of serial readings of the hæmagglutination titre is of obvious importance. It was therefore decided that serial examination of the serum in groups of medically and surgically treated patients might throw some light on the prognosis in these patients and the factors influencing the reaction. These are the results we wish to record.

Laboratory Technique

1. Sensitisation of red cells with tuberculin.

Fresh Group O rhesus-negative red cells were thrice washed in normal saline and resuspended in a 1 : 10 dilution of Lederle 4X Old Tuberculin, 1 part of packed cells to 10 parts of the diluted tuberculin. This mixture was incubated for one hour and shaken thoroughly at fifteen-minute intervals during this period. The cells were again washed three times in normal saline and then made up as a 1 per cent. suspension in saline.

2. Treatment of sera.

The serum from freshly taken blood was separated from the clot and inactivated at 56° C. for thirty minutes. It was stored before use at – 20° C. No sera were kept for longer than one week before examination.

3. Hæmagglutination test.

Doubling dilutions of the serum with saline in 7 by 50 mm. tubes were made with a range of 1 : 2 to 1 : 16. One volume of the suspended tuberculin

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treated cells was added to each tube. These were thoroughly mixed and incubated at 37° C. for two hours. They were then centrifuged for one minute at 1,500 r.p.m. and the button of red cells examined on a slide microscopically for the presence of agglutination.

The following controls were used:

(*a*) A washed suspension of untreated red cells with the patient's serum.
(*b*) A washed suspension of sensitised red cells with saline.
(*c*) A washed suspension of sensitised red cells with serum from non-tuberculous cases. One hundred and twenty non-tuberculous sera were tested during the period these investigations cover. Nine per cent. of these were positive.

When a test serum gave a positive agglutination in a dilution of 1 : 16 it was then titrated out to its end point.

In recording an increase or decrease in the titre only a " two tube " change was regarded as significant—*e.g.*, a change from 1 : 2 to 1 : 8. For this purpose the original and final titres were taken.

Results

Sera from a total of 111 patients have been serially examined for periods ranging from three to fourteen months. These cases may be divided into four groups:

Group 1. *Resection cases.*

Tests were performed before resection on twenty-five patients. Subsequent post-operative examinations were carried out at monthly intervals for three months. These patients were divided into three groups:

(*a*) Pneumonectomy (all specimens showed a destroyed lung).
(*b*) Lobectomy (specimens showed total destruction in four cases and partial destruction in ten cases).
(*c*) Segmentectomy.

This grouping was adopted to determine any relationship between the hæmagglutination titre and the amount of tuberculous tissue removed at operation. The results in this series are shown in Table I. Table II shows the relationship between the hæmagglutination titre and radiological evidence of residual disease after resection.

TABLE I.—EFFECT OF RESECTION ON THE HAEMAGGLUTINATION REACTION

Type of Resection	*Hæmagglutination Titre*		
	Lower	*Unchanged*	*Higher*
Pneumonectomy	1	6	—
Lobectomy (destroyed lobe)	—	4	—
Lobectomy (partially destroyed lobe)	1	8	1
Segmentectomy	1	3	—
Total (25)	3	21	1

84 per cent. of cases showed a positive reaction before resection (positive=1 : 8).

TABLE II.—RELATIONSHIP BETWEEN HÆMAGGLUTINATION REACTION AND RADIOLOGICAL EVIDENCE OF RESIDUAL DISEASE

Radiological Evidence of Residual Disease	*Hæmagglutination Titre*		
	Lower	*Unchanged*	*Higher*
No disease	2	13	—
Minimal infiltration (direct film negative) ..	1	6	1
Marked disease (direct film positive)	—	2	—
Total (25)	3	21	1

Group 2. Thoracoplasty cases.

Tests were carried out as for the resection cases on twenty-six patients before, and at monthly intervals for three months following, thoracoplasty. The results are shown in Table III.

TABLE III.—EFFECT OF THORACOPLASTY ON THE HÆMAGGLUTINATION REACTION

Result of Thoracoplasty	*Hæmagglutination Titre*		
	Lower	*Unchanged*	*Higher*
Successful	1	21	2
Unsuccessful	—	2	—
Total (26)	1	23	2

Successful=immediate cavity closure and sputum conversion. Unsuccessful=persistence of positive sputum with or without the presence of contralateral cavitation.
73 per cent. of cases showed a positive reaction before thoracoplasty (positive=1 : 8).

Group 3. Medically treated cases.

The hæmagglutination titre has been repeated after periods ranging from ten to fourteen months on twenty-eight medically treated cases. These twenty-eight form part of the original series of 130 cases reported from this hospital (Hinson, Richardson Jones and Chamberlin, 1952). The results are shown in Table IV.

TABLE IV.—EFFECT OF MEDICAL TREATMENT ON THE HÆMAGGLUTINATION REACTION

Clinical State	*Hæmagglutination Titre*		
	Lower	*Unchanged*	*Higher*
Improved	3	13	2
No change	2	5	1
Worse	—	2	—
Total (28)	5	20	3

71 per cent. of cases showed a positive reaction initially (positive=1 : 8).

Group 4. Isoniazid cases.

The serum of thirty-two patients was tested for its hæmagglutination titre before the administration of isoniazid. Further tests were performed at monthly intervals for three months during therapy (Table V).

TABLE V.—EFFECT OF ISONIAZID ON THE HÆMAGGLUTINATION REACTION

Hæmagglutination Titre		
Lower	*Unchanged*	*Higher*
1	29	2

75 per cent. of cases had a positive reaction before treatment (positive=1 : 8).

Discussion

In our series of resection cases the removal of varying amounts of tuberculous lung resulted in no change in the hæmagglutination titre in twenty-one out of twenty-five cases. It would appear that the antibodies demonstrated by the hæmagglutination reaction persist after the removal of large amounts of tuberculous tissue. For exactly how long they remain we cannot yet say.

Our results in twenty-six patients treated by thoracoplasty are essentially similar to the resection group. Twenty-one out of twenty-six cases showed no change in titre, although mechanically and bacteriologically the thoracoplasties were successful, and one might have anticipated a fall in titre following a permanent measure to stabilise the disease. Seventeen of these cases showed no radiological evidence of contralateral disease.

Twenty out of twenty-eight medically treated cases showed no change in titre, although thirteen were clinically very markedly improved and leading a normal life. These figures are in agreement with those of Spain, Childress and Rowe (1952), who found no change in titre in sixty-four out of eighty-nine tuberculous patients examined over a period of six to thirteen months.

Finally, treatment with isoniazid produced no change in titre in twenty-nine out of thirty-two cases. These patients were receiving no other antituberculous drugs during this period.

It is not yet clear as to the mechanism whereby these antibodies are formed. Middlebrook (1950) suggests that they are formed in response to products of lysis of the bacilli and that the release of antigen, stimulating production of the antibodies, ceases soon after the bacilli stop multiplying and spreading. Dubos (1950) has also put forward the view that hæmagglutinins represent a response to actively growing tubercle bacilli. Hinson *et al.* (1952) considered the possibility of the metabolic products from multiplying bacilli as an alternative source of antigen.

Tubercle bacilli are aerobic organisms. They are present in the lining of a cavity and the free availability of oxygen is a factor in their proliferation. Whether the antigen is lytic or metabolic in origin, multiplying bacilli as present in cavities may be assumed, from the views outlined above, to be a potent factor in the maintenance of the hæmagglutination titre. In support of this assumption is the fact that in our pneumonectomy group all cases had positive titres and all resected specimens showed cavitation. As stated previously, there has been no significant change in titre following resection.

The conclusion that might be drawn from these results is that the aforementioned hypotheses are untenable. However, minimal antigenic stimulus cannot be excluded. Nine of our twenty-five resection cases showed bronchial

tuberculosis at the site of surgical section and radiological absence of disease is not necessarily pathological proof.

A further point to be considered is the effect on the hæmagglutination titre of anti-tuberculous drugs. Gernez-Rieux and Tacquet (1950), using rabbits as experimental animals, reported a fall in titre following the administration of streptomycin. In man, Hinson *et al.* (1952) found no correlation between the hæmagglutination titre and the amount of streptomycin administered. In our group of patients treated with isoniazid the hæmagglutination titre was maintained (Table V).

The strains of tubercle bacilli isolated from these patients before treatment were all sensitive to this drug. Whether or not isoniazid is bactericidal or bacteriostatic, a diminution in the multiplying bacilli would be anticipated following its use.

We suggest that the antibodies detected in the hæmagglutination reaction reach a certain level and that this antibody pattern remains almost constant regardless of progression, excision or healing of disease, and we conclude from the results shown in Table IV that this test has no value in prognosis.

Summary

1. The hæmagglutination reaction is briefly discussed.
2. Serial reactions on medically and surgically treated cases are recorded.
3. No significant change was found in these groups of cases.

We wish to thank the Consultant Staff of the London Chest Hospital for permission to use their cases for these investigations.

We are grateful to Dr. K. F. W. Hinson for his help and advice in the preparation of this paper.

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MEDIASTINAL EMPHYSEMA COMPLICATING THERAPEUTIC PNEUMOPERITONEUM

By C. M. Tinker

From the Chest Clinic, St. Helier's Hospital, Surrey

Introduction

Mediastinal emphysema complicating therapeutic pneumoperitoneum is a rare occurrence, the mechanism of which has not been satisfactorily demonstrated. Only four cases have been recorded in the English literature, and the purpose of this paper is to record a further case and discuss the etiology.

Pneumoperitoneum as a cause of mediastinal emphysema received a passing mention as early as 1927, in a paper by Jehn and Nissen on the pathology and clinical features of this condition. The pneumoperitoneum in this case was presumably a diagnostic one, as therapeutic pneumoperitoneum did not come into general use until 1934, when Banyai reported a series of 100 cases treated by this means. In 1939 Banyai and Jurgens described seven cases of mediastinal emphysema following pneumoperitoneum refills given by the intercostal route. In these cases Banyai thought it possible that the needle had damaged the diaphragm, and that the air forced back into the track had stripped the peritoneum and reached the mediastimum. The problem differs from the one about to be discussed, when the air was injected by the para-umbilical route.

In 1950 Lemanissier, Breant and Fourchon described in detail two cases of mediastinal emphysema following pneumoperitoneum refills given by the para-umbilical route. In the first case the incident occurred eight days after a normal refill, and four months after the induction of the pneumoperitoneum. It was ascribed to severe vomiting in an attack of gastro-enteritis. The clinical signs were confirmed by radiography. In the second case the incident occurred five months after induction, and immediately after a weekly refill in which the pressures were noted as higher than usual The clinical diagnosis was confirmed by radiography, and it was suggested that an extra-peritoneal refill had inadvertently been given. The authors comment that both patients were portly women in whom lax abdominal musculature and readily detachable peritoneum might be expected.

They conclude, therefore, that there are two possible explanations of this event when refills are given by the para-umbilical route:

(1) Gross rise in the intraperitoneal pressure caused by a very large pneumoperitoneum, severe muscular effort, etc., causes a local rupture of the peritoneum opposite a feeble portion of the abdominal wall; particularly the anterior insertion of the diaphragm.

(2) An extraperitoneal refill strips the peritoneum, and forms an extra-peritoneal air pocket. This permits the air to track through into the mediastinum.

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Case Report

In the case now to be recorded, the clinical picture was similar.

G.P., aged 22, a married woman with one child. A pneumoperitoneum had been induced eighteen months previously and maintained by fortnightly refills of 1,200 c.c. of air, the final pressures ranging from +6 to +16 cm. of water. The left phrenic nerve had been crushed and had recovered some months previously.

On this occasion the needle was inserted at the outer margin of the left rectus muscle, on a level with the umbilicus. The anterior abdominal wall was tightly stretched, as the patient had omitted to remove the pillow provided for the previous pneumothorax refill. Eight hundred c.c. of air was introduced, the final pressure reading being +20 to +25. The unusually high pressure was attributed to the position in which the patient was lying. During the refill the patient complained of tightness in the epigastrium, and then behind the sternum and in the back. She sat on a chair while the next patient was refilled, and then remarked that she had swelling in the neck. Inspection revealed large blebs of surgical emphysema occupying the right supraclavicular fossa and supra-sternal notch. There was no dyspnœa or dysphonia, and no evidence of pneumothorax. X-rays were taken, and by the time the films were developed the patient appeared to have fully recovered. The following week the patient presented herself for refill as usual. She stated that pain had recurred with severity the following day, and she had remained in bed. She had had discomfort most of the week. Screening did not reveal any obvious air in the mediastinum, and the pneumoperitoneum appeared much as usual (Fig. 1). A further refill of 800 c.c. was given, and no complaint was made. Since then fortnightly refills of 1,200 c.c. have been continued without incident.

Discussion

It is thought that in this case the air was probably delivered into the peritoneum, since there was a good respiratory swing of +20+25, and no air was visible in anterior or posterior abdominal walls. If so, the high pressure at the time of the refill must have been responsible for forcing air through a weak segment of the diaphragm. Such vulnerable areas exist at the point where vessels, nerves, and œsophagus traverse the diaphragm, and at the sites of fusion of embryonic diaphragmatic elements. In this case it is suggested that air ruptured the peritoneum between the sternal and costal attachments of the diaphragm and tracked up behind the sternum. The lateral X-ray shows a column of air separating the heart from the sternum (Fig. 2). No attempt was made to remove the pneumoperitoneum, and the condition apparently recurred during the following twenty-four hours. Barium meal at a later date excluded any para-œsophageal herniation.

The incrimination of high intraperitoneal pressures as a cause of mediastinal emphysema is in line with the conclusions of other authors regarding the increasing incidence of spontaneous pneumothorax in pneumoperitoneum. Though some of the recorded cases of spontaneous pneumothorax occurred shortly after the induction of pneumoperitoneum, and could be attributed to congenital defects in the diaphragm (Newlyn Smith, 1943; and Sita-Lumsden, 1949; etc.), recently a number of cases have been recorded which have occurred after a successful pneumoperitoneum has been maintained for some months

(Spencer Jones and Yuill, 1952; Partington, 1952—personal communication), and the mishap has been attributed to rupture of the diaphragm. Blebs have been observed on the upper surface of the diaphragm at thoracoscopy (Laird, 1945; Repa and Jacobson, 1951), and a lesion resembling organising blood clot was seen in the case of Wynn-Williams (1951) and Spencer Jones and Yuill (1952), which was thought to be the actual site of the rupture.

Diagnosis

A minor degree of mediastinal emphysema is easily overlooked. The diagnosis should be considered when a patient complains of substernal discomfort, tightness, or pain, associated with pain between the shoulder blades. There is usually little radiation. Stiffness or discomfort in the neck may be noted. The following signs should be sought:

(*a*) Absence of præcordial dullness to percussion.

(*b*) Hamman's sign. On auscultation over the præcordium crackling crepitations are heard. These may be synchronous with the heart-beat, and are then described as a " systolic crunch " by Hamman. In some cases this may be so loud as to be clearly audible from the foot of the bed, and may occur in cases of spontaneous mediastinal emphysema (Hamman), though more familiar in association with trauma.

(*c*) Air in the subcutaneous tissues of the neck.

(*d*) X-rays show the mediastinal pleura, pushed laterally by air in the mediastinum, appearing as a thin detached line on either side of the mediastinum (Fig. 1). In the lateral film a column of air separates the heart from the sternum (Fig. 2).

Summary

A case of mediastinal emphysema following a pneumoperitoneum refill is described, and attributed to high intra-abdominal pressure.

My acknowledgments are due to Dr. Frank Harwood and Dr. Hayden Partington for their interest and encouragement in the preparation of this paper, and to Dr. Brooks for permission to publish.

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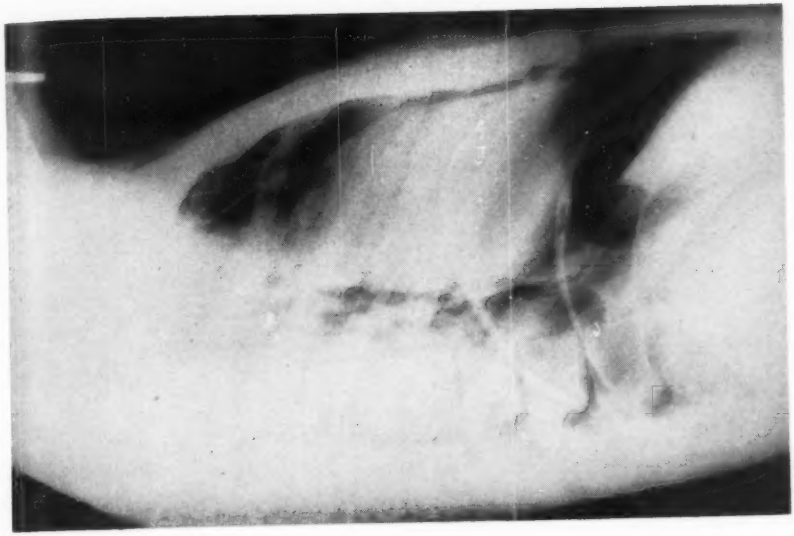

FIG. 2.—Lateral X-ray showing a column of air separating the heart from the mediastinum.

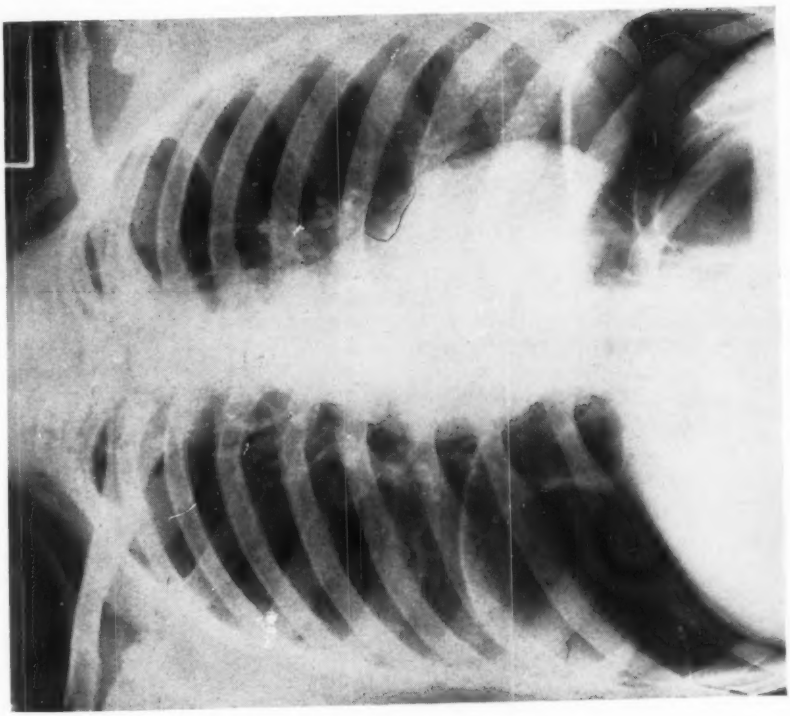

FIG. 1.—P.A. View showing a normal pneumoperitoneum with some air in the mediastinum appearing as a thin detached line on either side of the mediastinum.

To face p. 48

AN EARLY TREATMENT CENTRE FOR PULMONARY TUBERCULOSIS

By F. E. de W. Cayley, Eric Sanders and Donald McIntosh
From Bevendean Hospital, Brighton

One of the problems in the treatment of pulmonary tuberculosis is the organisation of methods whereby early cases amenable to treatment are prevented from becoming advanced while waiting for a bed in the conventional sanatorium.

This has been solved in many areas by schemes for domiciliary active treatment.

A common feature of such schemes is the initiation of collapse therapy in the patient's home, and the Chest Physician in charge of the Clinic has a few beds under his care to which he can admit cases for a short period for minor surgery or the treatment of complications.

In consequence much work devolves on the already overworked Chest Clinic (Heller, 1949).

In the area of the South-east Regional Hospital Board it was thought preferable to set up early treatment centres where proper diet and environment could be provided with enforced rest, where the administration of drugs could be supervised, where activity could be better assessed, and where patients could be taught the sanatorium régime.

The aim was to retain patients in hospital for only three or four months and then to discharge them for follow-up by the Chest Clinics or in a few cases to transfer them to a sanatorium if the home conditions were unsuitable.

The following is an account of the first year's working at such a centre. The hospital was first opened for this purpose at the beginning of 1951 with 56 beds but, as more nurses were recruited, the number was slowly increased to 141.

312 patients were discharged in the first year. Of these 233 fell into the category of early cases. The remaining 79 consisted of 25 cases admitted as emergencies, 33 admitted for a few nights for a minor operation such as phrenic crush or bronchoscopy, 12 children with primary tuberculosis, and 3 patients took their own discharge before treatment could be started. In addition, 6 cases sent in as early tuberculosis proved to have a non-tuberculous condition.

For the early cases the average length of stay was 118 days.

Contact History.—There was no history of contact in 131 cases, 70 gave a history of one close relative having had tuberculosis, 26 of two relatives, 4 gave a history of three relatives with tuberculosis and 2 incriminated a lodger as the cause of their infection.

Symptoms.—61 had no symptoms referable to the chest but had been discovered at routine or Mass X-ray examination—46 at Mass X-ray, 8 on call-up to the Forces and 7 when having a barium meal. The remainder had either local or constitutional symptoms suggestive of pulmonary tuberculosis; 21 of these found the Mass X-ray Units the most convenient way of getting their first X-ray.

(*Received for publication October 24, 1952.*)

Sex.—There were 86 men and 147 women, because more beds were available for females than males.

Age.—The main age group was 15 to 40, with the peak in the 20 to 25 age group.

TABLE I.—AGE GROUPS AND SEX

Age:	*Under* 15	15 *to* 25	25 *to* 35	35 *to* 45	45 *to* 55	55 *to* 65	*Total*
Male	3	34	29	16	3	1	86
Female	3	74	44	22	3	1	147
Total	6	108	73	38	6	2	233

Sputum.—168 never had a positive sputum; 33 had a positive smear on admission and became negative; 20 were positive on smear before admission and were negative on admission; 8 were still positive on discharge but were awaiting major surgery; 4 had a positive culture on admission and converted to negative.

Treatment before Admission.—42 had no treatment before admission; the remainder had bed-rest with or without specific drug therapy as shown in Table II.

TABLE II.—TREATMENT BEFORE ADMISSION

Months' Treatment:	*I*	*II*	*III*	*IV*	*V*	*VI*	*VII*	*VIII*	*IX*	*X*	*XI*	*XII or more*	*Total*
Bed	41	24	16	10	8	8	2	0	3	1	1	3	117
Bed and P.A.S.	11	5	8	3	2	2	0	1	0	0	0	0	32
Bed P.A.S. and streptomycin ..	13	7	7	4	1	1	1	0	1	0	0	0	35

Previous San. No immediate treatment: 3.
P.A.S., Streptomycin 30 days. No Bed: 4.
No treatment: 42.

It will be observed that 4 cases had a course of P.A.S. and streptomycin without being put to bed.

X-ray Appearances.—Some attempt has been made to analyse these in Tables III, IV and V. It was seen that 132 had cavities, 163 were unilateral, 145 had only one zone affected.

TABLE III.—X-RAY APPEARANCES

Right side ..	78
Left side ..	85
Bilateral ..	70

TABLE IV.—X-RAY APPEARANCES

Zones	
Upper only ..	164
Mid only ..	26
Lower only ..	6
Mixed ..	37

TABLE V.—X-RAY APPEARANCES

Total Zones	
One ..	145
Two ..	78
Three ..	4
Four ..	6

Diagnosis.—Where the sputum was negative, this was based on the appearance in serial X-rays of infiltration of a tuberculous type with or without cavitation, which did not clear up or alter appreciably on watching for at least two months. Additional pointers were obtained from the presence of calcification, a positive Mantoux test, contact history and the youth of the patient.

Period of Assessment before starting Treatment.—This naturally varied with the length of observation by the Chest Clinic while waiting for a bed, and with the severity and nature of the case. It was thought that no A.P. should be induced until the patient had been on bed-rest for a month either at home or in hospital.

Activity of the Lesion.—Temperature and sedimentation rate are quite often normal in these early cases; the need for treatment was decided more upon the softness and extent of the chest lesion. Tomographic proof of cavitation or a lesion increasing in size in spite of bed-rest obviously required active treatment. In doubtful cases the background of the patient sometimes swayed the balance in favour of collapse therapy; for example, a bad family history, a bad home or strenuous employment.

The Decision to use P.A.S. and Streptomycin.—In non-A.P. cases it was used for predominantly infiltrative lesions of recent origin and fairly considerable extent, and where constitutional symptoms were marked. In A.P. cases the questions were asked: Is there an increased risk of effusion if streptomycin is withheld? Could this lesion, which is too active for an A.P., become suitable after treatment by streptomycin for a month or two? Would it be better to withhold streptomycin now, so that it is still effective if a major operation is needed subsequently? The decision was made according to the replies to these questions.

Treatment.—Bed-rest was the only form of treatment in 43 cases. These were the cases in which there was doubt as to whether the lesion was a primary one: in cases of bilateral apical minimal disease in which bilateral A.P.s seemed too drastic a treatment for such small lesions, in cases of doubtful activity, and in those cases where the month's preliminary bed-rest had produced marked resolution of the lesion.

A period of three months was the minimum; this included the time in bed before admission. Some were sent out to be looked after in bed at home by relatives.

P.A.S. and streptomycin were given to 69. In 50 it was used as a preparation for an A.P., in 2 for a thoracoplasty; in the remaining 17 it was the only form of treatment apart from bed-rest. The normal course lasted three months; continuous daily injections of 1 gramme were given.

Induction of artificial pneumothorax was attempted in 161 cases. 89 unilateral and 7 bilateral cases were completely uneventful and satisfactory. A further 23 cases were discharged with an A.P. and are commented on below. In 42 the A.P. was unsuccessful. Thus a total of 119 were discharged with an A.P.

Of the 42 unsuccessful ones, 13 had no space; in 22 the adhesions were indivisible; 4 developed an effusion, 1 after a hæmothorax following adhesion section; and in 3 the A.P. was contraselective. Further treatment was advised in 17 of the failures.

Of the 23 cases who were discharged with an A.P. but on whom comment is

required, 2 were awaiting surgery on the other side; 3 had a bilateral A.P. attempted, but only on one side was it successful, although on review this side had improved; 14 were maintained in spite of a few indivisible adhesions; 3 had a severe hæmothorax after adhesion section; 1 had a transient pleural effusion.

Hæmothorax.—4 cases of severe hæmothorax occurred after adhesion section; 2 were given blood transfusions. They were treated by aspiration, but in spite of energetic treatment 2 developed pleural thickening and 1 had to be abandoned owing to subsequent development of an effusion.

Phrenic Crush and Artificial Pneumoperitoneum.—In 1 patient this was the only treatment apart from rest; 5 of the failed A.P.s also had this form of treatment.

Thoracoplasty or a similar operation was recommended in 21 cases; 7 of these were primary, 12 were secondary to a failed A.P. and 2 were on the contralateral side to an A.P.

Primary lobectomy was recommended in 3 cases; in 2 of these the diagnosis was tuberculosis of a collapsed and bronchiectatic lower lobe, which was confirmed in the assessment period by bronchogram.

One patient was mobilised and discharged after assessment as being inactive.

Mobilisation after Treatment.—Patients were usually discharged when they were up for two hours unless there was no one to look after them at home. After thoracoscopy they were only kept in bed for two weeks and thereafter their time up was increased by half an hour each week unless there was a special reason for going slow.

Follow-up.—We are grateful to the Chest Physicians at the Clinics for reports which enabled us to get some idea of the progress of these patients after leaving the hospital.

At the time of the follow-up some had been discharged twelve months and others only four months.

Degree of Activity.—At the time of the follow-up, 93 were working, 45 were up all day but not working, 38 were up six to eight hours, 50 were still in bed or in a sanatorium, six had moved, 1 had died.

Of the 85 patients discharged or transferred without any form of collapse therapy and who were not waiting for major surgery, 34 were working, 11 were up all day, 15 up six to eight hours, 2 were still on bed-rest at home, 8 had spread. Of these 4 had been admitted to a sanatorium and 1 had had a lobectomy, 4 had moved and 11 patients were still in the sanatorium to which they had been transferred.

A.P. Cases.—Of the 119 patients who had been discharged with an A.P., 51 were working, 33 were up all day, 16 were up six to eight hours, 2 were still in bed because of contralateral disease, 6 had had their A.P. abandoned for various reasons given below, 6 had had some setback which required further treatment, 1 patient only had been transferred to a sanatorium because of bad home conditions and was still there, 1 was pregnant, 1 had moved, and 2 were awaiting surgery on the other side.

The reasons for abandoning the A.P. in the 6 cases were : fluid in 2—1 up eight hours, 1 working; obliterative pleurisy in 1, who was working; 3 had been already deliberately abandoned, 1 of whom recavitated and who had to be reinduced, successfully, I am glad to say.

The setbacks which required further treatment in 6 were: spread to the other side in 3; serous effusion in 2 which necessitated aspirations before they could be abandoned; 1 had reopened his cavity in spite of an adequate A.P.—he had a lobectomy and died shortly after the operation from thrombosis in the internal carotid.

P.P. Cases.—3 were working; 3 up for six to eight hours; 1 had moved.

Surgical Cases.—7 were waiting in the surgical sanatorium, 1 was waiting at home; 6 had had a thoracoplasty; 3 a plomb with plastic balls; 3 a lobectomy; 4 had closed their cavities in the waiting period—1 working, 1 up all day, 2 up eight hours.

Discussion

Thanks to a fairly high rate of turnover our waiting list for female patients has been reduced to two weeks and we are now able, in our second year, to admit more advanced cases who require longer in-patient treatment. Males still have to wait two months, but this is a good deal shorter than formerly.

It will be obvious that the cases admitted were a highly selected group.

It is, we know, contrary to the prevailing fashion to induce a large number of A.P.s; but so far none of our A.P. cases has come to any great permanent harm. Most of our cases were not of the type usually associated with surgery. Bed-rest by itself probably gives about 20 per cent. less security against relapse than an A.P. and requires a much longer period away from work.

Moreover, the facilities for thoracic surgery in the region are not yet able to deal even with the cases where the need for operation is unambiguous. Had better facilities for thoracic surgery been available, it is probable that a few more of our cases would have been operated on, notably the cavitating tuberculoma, but even in these an A.P. sometimes closes a cavity.

We are of the opinion that, in the treatment of pulmonary tuberculosis, the simpler and safer methods should be, if possible, tried first; and when they fail it is the time to consider an operation.

We fear, in the present craze for resection, that as the years go by an appreciable number will develop fresh lesions in the remaining lobes; the mortality rate for resection is about 4 per cent. and the post-operative period is not without its anxieties.

Thoracoplasty is not usually undertaken immediately after the diagnosis of pulmonary tuberculosis has been made, and between the tentative decision to put a patient on the waiting list and a bed becoming available simpler methods may render the operation unnecessary.

Effusions.—No case of empyema has yet developed. 5 developed effusions while in hospital, 10 in the follow-up period. In 8 the effusion has necessitated the abandonment of the A.P., but in the meantime the lung lesion has improved.

Atelectasis.—Atelectasis without cavitation in the diseased upper lobe was present on discharge in 8 cases; 6 on follow-up have re-aerated; 1 abandoned owing to fluid is working; 1 who also had indivisible adhesions running to the subclavian artery still has atelectasis and is susceptible to bronchitis and cough. It is probable that she ought to have a lobectomy.

Undivided Adhesions.—14 A.P.s were maintained in spite of a few indivisible adhesions. In each case the advice of the surgeon doing the thoracoscopy was sought as to the number and risk of their snapping. A further X-ray was examined for the degree of relaxation of the lesion before the final decision to continue was made. 11 of these are satisfactory. 1 has obliterated but is up all day. 1, who had bilateral A.P.s, one side free, the other side with a few indivisible adhesions, has some fluid on both sides; but both A.P.s are being maintained and the patient is up seven hours. The other is the patient mentioned in the previous paragraph as having in addition atelectasis.

Prevention.—If B.C.G. is going to prove efficacious in this country, since half of our cases were family contacts, the incidence in this group may be appreciably lowered in the future generation.

Time in Bed after Thoracoscopy.—It has not been our impression that the short period in bed after thoracoscopy has been followed by a greater incidence of effusion.

Treatment after Discharge.—The standard of after-care has been extremely high and there is only a small variation between one Clinic and another as to the rate of mobilisation and return to work. The three A.P.s that have already been abandoned deliberately have, perhaps, been given up rather soon, as was borne out by the case that had to be reinduced.

Administrative Comments.—In spite of the slow deliberation of a host of committees, the division of administration into three strata (Region, Group and Hospital), and restraint on expenditure, with goodwill all round most difficulties have been smoothed out extremely quickly.

Nursing.—Recruiting of staff has so far proved easier than expected, but the scheme for secondment from a General Hospital suggested by the General Nursing Council has not met with local favour, partly, we think, because the General Hospitals themselves have their own recruiting difficulties, partly because the nurses have a fear of contracting the disease, and partly because the Chest Hospital itself fears to lose its individuality if submerged by a larger hospital. Fortunately, the Diploma of the British Tuberculosis Association has proved some inducement to the prospective nurse.

Conclusion

The setting up of these early treatment centres has helped considerably in the solution of the regional tuberculosis problem, and a large proportion of the patients seem to have done quite well.

We would like to express our thanks to Mr. A. H. M. Siddons, M.R.C.P., F.R.C.S., for his ever-helpful surgical advice and to his indefatigable team of Registrars who have rendered us such valuable assistance.

REFERENCE

HELLER, R. (1949): *Tubercle*, **30**, 204; *Lancet*, 1952, **i**, 305.

REVIEWS OF BOOKS

Medical Biographies. By PHILIP MARSHALL DALE, M.D. University of Oklahoma Press, Norman, U.S.A. Pp. 259. 32 portraits. Price $4.

To the books and articles on the illnesses and deaths of the famous (and infamous) Dr. Dale's is a worthy contribution.

Its thirty-three subjects extend from Buddha, 563 B.C., to Robert Louis Stevenson, 1894. In some cases the enthusiasm of the historian predominates and the presentation is mainly biographical; in other cases, interest is chiefly related to pathology; and in others again both are combined, especially when it is desired to represent the influence of disease upon character and activity and to speculate upon the effect that modern therapy might have had upon the history of the world.

Thus the old Maxwell Mackenzie controversy. A correct diagnosis of malignant disease at an early stage might have prevented the tragic consequences of the premature demise of the Emperor Frederick III of Prussia. Had this been averted, so that he would have been on the throne during the fateful first fourteen years of the twentieth century, the recent course of world history might well have been a happier one.

Henry VIII is the favourite among historians, who find an opportunity to indulge their prejudices in discussing that much maligned, perhaps much misunderstood monarch. In opposition to Sir Arthur MacNalty in his recent book, Dr. Dale accepts the influence of syphilis. Incidentally, it requires little ingenuity in the straining of probabilities to incriminate the *Treponema pallidum* for the physical and psychiatric consequences in many other instances. One may suggest to an appropriate writer as an attractive subject " The Influence of Syphilis upon World History "—if indeed such a work has not already appeared.

ADOLPHE ABRAHAMS

Help Yourself Get Well. By MARJORIE MCDONALD PYLE, M.D. Heinemann. 12s. 6d.

This book, written by a doctor herself a victim of pulmonary tuberculosis, appears to be of American origin, but the information about English Tuberculosis Schemes and facilities has been incorporated in it, so that it can be of equal use to readers in this country. The author is undoubtedly imbued with a spirit of enthusiasm to help those suffering from pulmonary tuberculosis, and although the contents of the book are pitched on a somewhat emotional key, it can be said that she succeeds in her task. For the same reason the book is, perhaps, rather a counsel of perfection which most patients would be unable to live up to, but having read it they have no excuse for pleading ignorance. Much of the information is normally acquired by patients in sanatoria as a result of familiarising themselves with the ordinary routine, which in many sanatoria is reinforced by lectures over the internal wireless system by members of the medical staff. It is to be doubted whether some of the views expressed in the chapter on " The Psychology of the Tuberculous " would find general acceptance, and in the chapter " We're in Good Company " a large number of names of famous sufferers from tuberculosis are given, going back as far as Mozart. One could perhaps doubt whether diagnosis in those days was

accurate enough to place some of the great names mentioned in a group of those suffering from tuberculosis.

These, however, are minor criticisms, and the book can be generally recommended as a useful guide to patients, although its style would seem to appeal more particularly to the feminine reader. One last remark—could not the title, for the benefit of English readers, be altered to " Help Yourself *to* Get Well?"

W. E. SNELL.

Modern Electrocardiography. By EUGENE LEPESCHKIN, M.D. London: Baillière, Tindall and Cox, Ltd. 1951. Pp. i-xiv+1-598; illustrated. Price 91s. 6d.).

Although only one volume has yet been published, the reviewer is led to believe that the volumes will constitute the most noteworthy work on electrocardiography since Lewis's classical work " The Mechanism and Graphic Registration of the Heart Beat." The first volume is devoted to disorders associated with a regular rhythm and the final volume will be concerned with the cardiac arrhythmias.

The book gives an accurate and detailed account of modern electrocardiography since 1933, when Frank Wilson began to eliminate much of the empiricism from clinical cardiography. Some idea of the scope of this work can be gained from knowing that the author numbers about ten thousand references and actually gives many more than this, as he often includes several references under one index number. Surprisingly he makes no reference to the work of W. H. Craib.

Both the theory and practice of cardiography are dealt with fully—a mastery of the second and third chapters, which summarise much of modern electrocardiographic theory, will make intelligible to the beginner most of the present-day literature, although the book is not really designed for the beginner but is essentially supplementary to existing textbooks. Throughout, Bernstein's membrane theory of electrical conductivity has been accepted.

The practical side of electrocardiography is not neglected; details of the various machines available are briefly given and few experienced cardiographers will fail to find a tip or two on how to eliminate A.C. interference.

To readers mostly interested in chest diseases the sections on pericarditis, pulmonary embolism, pulmonary heart disease and right ventricular hypertrophy and strain will perhaps appeal most. All are done excellently.

On the debit side a few minor criticisms: the misprints are fairly numerous even for such a detailed work and one or two are actually misleading. For example, on p. 63 each of Bayley's sextants is given as 30°, whereas they are of course 60°, and his name is spelt incorrectly in the legend to Fig. 17. On p. 75 Fig. 27b should read 23b and on p. 95 Fig. 13 should read Fig. 28. A particularly irritating misprint which will certainly puzzle beginners trying to understand Fig. 38 occurs on p. 118, where ABF should read ABG.

SAMUEL ORAM.

The St. Thomas's Reports, Second Series. Vol. VII, 1951. Price 10s. 6d.

It is not likely that all the eighteen articles comprised in the present series will appeal to every reader. But it is equally unlikely that any one reader will fail to be attracted by something in his particular field. The analysis of 1,000 barium meal examinations, whilst admitting a high degree of accuracy in the diagnosis of gastric and duodenal ulceration and of proliferative neoplasms of the stomach, appends the admission that the early diagnosis of malignant

disease has been particularly unsuccessful. Bacterial endocarditis in a child of two and a half years is described with the successful treatment by aureomycin after the development of penicillin resistance. The difficulty of distinguishing cancer of the lung from chronic pneumonia is well represented in a valuable article by Mr. Meredith Brown, who describes the cure by thoracotomy in eight cases where the more baleful diagnosis had been made on clinical and radiological grounds. Other articles of strong general appeal are the treatment of acute hæmorrhage from peptic ulcer in men, and iron deficiency anæmia. The latter is exceptionally informative in its consideration of the influence of achlorhydria and the significance of koilonychia. Twenty-two cases are recorded—all in women, of whom no fewer than fifteen were in the fifth decade. It would be interesting to have a subsequent article on the subject of such anæmia in female adolescents and in men. Instances are rare, but they have occurred in the reviewer's experience.

ADOLPHE ABRAHAMS

Brompton Hospital Reports. Vol. XX, 1951. Pp. 196+vii. Price 12s. 6d.

ONCE again the editors are to be congratulated on this beautifully produced volume. Fourteen of the fifteen papers comprising the volume have already appeared in the various medical journals. The book opens with the Goulstonian Lectures on "Suppurative Pneumonia." These are models of their kind and have already made their mark as a valuable contribution to the subject.

The report on the first five years' work of the Joint Consultation Clinic for Neoplastic Diseases of the Brompton and Royal Cancer Hospitals on Carcinoma of the Bronchus is a valuable and authoritative contribution on the subject.

The diagrams of the bronchial tree with corresponding bronchograms have been prepared primarily for use in the Institute of Diseases of the Chest and have not previously been published in this form. Incorporating the new nomenclature as adopted by the Thoracic Society and the new numbering, they should prove very helpful for teaching purposes.

Other contributions include a review of Tuberculosis of the Upper Air Passages and its Treatment, by F. C. Ormerod; Tuberculomata of the Lung, by E. N. Moyes; Intrapleural Hæmorrhage in Artificial Pneumothorax, by J. T. Harold; and the Brompton Rehabilitation Clinic, by N. C. Oswald.

PHILIP ELLMAN

The Protection of the Nurse against Tuberculosis. By F. A. H. SIMMONDS. National Association for the Prevention of Tuberculosis. Pp. 90. Price 10s. 6d.

The purpose of this little volume is self-explanatory, and, as pointed out in the Foreword, it is primarily intended for medical superintendents, matrons, nurse teachers and home sisters. Although all the measures recommended are already familiar to those whose sole or main duties lie in sanatoria or tuberculosis wards, their reiteration and wider publication are desirable at a time when increasing numbers of tuberculosis cases are being treated in general hospitals and when shortages of staff often result in such cases being nursed by those unaware of the dangers and problems involved.

Many of the recommendations are of general application, such as the selection of student nurses, provision of suitable living accomodation and a balanced diet, and the arrangement of proper working hours, leisure and rest. Attention to these details is of equal importance to nurses in general and to the population at large in order to increase their resistance to infection, particularly by the tubercle bacillus. The sections which deal more specifically

with tuberculosis cover all the important points and are simply and lucidly written. The account of the tuberculin reaction and its significance, particularly in relation to B.C.G. vaccination and the primary infection, should be helpful to those responsible for selecting trainee nurses and the care of their health subsequently. The details of nursing technique, especially those concerning disposal of sputum and sterilisation of fomites, should already be thoroughly familiar to those in charge of tuberculosis and infectious wards, but cannot be overstressed. The recommendations that all Mantoux-negative nurses should be vaccinated with B.C.G. and that all patients entering a general hospital should have routine chest X-rays will meet with general approval.

The simple style of this book and its small size make it a suitable handbook for nurses, and it could be read with profit by lay and medical authorities concerned with the problems of tuberculosis.

J. H. P. JOHNSON.

BOOKS RECEIVED

Diseases of the Chest. By Sir Geoffrey Marshall and Kenneth Perry. Butterworth. 1952. In two volumes: I, Pp. 456+xi+19. II, Pp. 413+vii+31. Price £7 7s. the set.

Tuberculosis Index and Abstracts of Current Literature. N.A.P.T. September 1952. Vol. 7, No. 3. Price 25s. a year.

Incidence of Pneumonia in Two Communities in New York State. By Doris Toucher. Milbank Memorial Fund, 40, Wall Street, New York 5. Pp. 15.

Change with Age in Susceptibility to Minor Respiratory Illness. By Jean Downes. Milbank Memorial Fund, 40, Wall Street, New York 5. Pp. 13.

What Everyone should Know about Tuberculosis. By M. Santosham. Crystal Publishing House, Madras. 1952. Pp. 239+xi. Price Rs. 6-8-0.

Diseases of the Chest. By T. Royle Dawber and Lloyd E. Hawes. Baillière, Tindall and Cox. Pp. 458. Price 76s. 6d.

The National Health Service in Great Britain. By Sir James Stirling Ross. Oxford University Press. Pp. 398. Price 30s.

Annales del Instituto de Farmacologia Española Vol. 1. Madrid. 1952. Pp. 336.

REPORTS

THE HEALTH SERVICE IN RETROSPECT

Ministry of Health's Annual Report, 1951*

Among the observations made in this Report, with special reference to tuberculosis, are the following:

"Shortage of beds for in-patient treatment of respiratory tuberculosis, mainly through lack of nursing staff, remains the chief problem of the tuberculosis service, but by the end of the period the situation had much improved. By setting aside for tuberculosis treatment suitable blocks in general and isolation hospitals, as well as by reopening closed beds as the nursing staff increased, 3,650 additional beds were made available between July 1950, and December 1951, bringing the total increase since the National Health Service began to over 6,000. The tuberculosis nursing staff increased from 5,337 whole-time and 1,028 part-time in July 1950 to 5,760 and 1,188 respectively at the end of 1951. The waiting list for in-patient treatment (which represents patients for whom a bed is not found within ten days of the date when the patient's name is put down for admission) fell from approximately 11,000 in December 1949 to about 7,000 in December 1951. What is more, the length of time taken to find beds for patients on the waiting list was generally much less at the end of the period than at the beginning of it, so there was substantially more improvement in the situation than is shown by the numerical reduction of the waiting list considered alone.

Mass miniature radiography continued to progress. By the end of 1951 there were 58 units working among the civil population, compared with 54 at the end of 1950 and 36 in 1948, and they had examined over 8,000,000 people."

PROGRESS AND HOPE IN TUBERCULOSIS

Annual Report of the National Association for the Prevention of Tuberculosis of Great Britain, 1951-1952.

Many of the Association's activities have been noted in our columns during the past year, but the Annual Report surveys its educational, research and propaganda efforts in the fight against tuberculosis through the whole of 1951-2. Its work extends to refresher courses, post-graduate lectures, scholarships for nurses, etc. The new policy of dividing the membership of the N.A.P.T. into sections is proving successful. Among these are the Medico-social, Sanatorium Matrons', Nurse Teachers' and Tuberculosis Nurses' Sections. Art therapy has proved a permanent and valuable activity of the N.A.P.T., while Rehabilitation Research, with special reference to home studies, has received special attention.

Publications of the N.A.P.T. are widespread and most have been reviewed in this Journal, notably the Bulletin which appears six times a year, the quarterly "Tuberculosis Index" containing abstracts of current literature, and the quarterly "Health Horizon" which seeks to place tuberculosis in proper focus against the background of general medicine and hygiene. Non-

* Report of the Ministry of Health (Part 1) April 1, 1950, to December 31, 1951. Cmd. No. 8655, H.M.S.O. Price 5s.

pulmonary Tuberculosis of Bovine Origin, The Protection of the Nurse against Tuberculosis, " Home Work " with a Difference, and the Handbook of Tuberculosis Activities have also been or will be noted in the Journal.

Finally, there are records of the work of the Tuberculosis Educational Institute, the Commonwealth Tuberculosis Associations and the contacts with these associations, and the inauguration of the scheme for the treatment of British children in Denmark.

N.A.P.T. HANDBOOK OF TUBERCULOSIS ACTIVITIES IN GREAT BRITAIN AND THE COMMONWEALTH

We have received the fully revised fourteenth edition of this handbook, which is a complete and comprehensive directory of tuberculosis and chest hospitals, sanatoria and chest clinics, Regional Hospital Boards, Hospital Management Committees and the like. It is a useful and almost encyclopædic volume of 425 pages, comprising the names of the medical staff of these institutions. There is a new and enlarged section which concerns the Local Authorities and their Medical Officers of Health, together with Divisional Medical Officers and statutory and voluntary Care Committees. The book is an invaluable compendium on tuberculosis activities in Great Britain and the Commonwealth, and is strongly recommended to all tuberculosis workers.

Annual Report of Derbyshire County Medical Officer of Health.

Dr. J. B. S. Morgan, County Medical Officer of Health, in the course of his Annual Report for 1951 observes that the emphasis under the National Heatlh Service is on curative medicine. He suggests that we require a reorientation of medicine so that prevention would be in the ascendant, in precisely the same way as in the police force, because it is obviously better that disease should be prevented than that the " detection and punishment " of offending micro-organisms should be effected after they have succeeded in committing the crime of disease !

The Report features observations from Chest Physicians and Medical Directors of Mass Radiography Units. Dr. Morgan notes that the scheme for the prevention and after-care of patients suffering from tuberculosis has continued to operate on well-established lines. Under the National Health Service Acts, District Medical Officers of Health are required to forward to the County Medical Officer of Health copies of notifications of infectious disease, including tuberculosis. From this information a register of all cases in the county is kept in the Central Office. Health Visitors are informed each week of all new cases, so that they may visit and give appropriate advice to the patient and relatives. Particulars of all notified cases are also forwarded to the Chest Physicians with a view to (i) arrangements being made for the treatment of patients; and (ii) their care in the community while awaiting admission to sanatoria. Regarding (ii), the Chest Physicians' recommendations are accepted concerning any services that come within the range of the authority's " Care and After-Care " scheme. Chest Physicians inform the department of new cases attending, and of patients removed from the register as having recovered from the disease, as having left the district, and so on, and this information, in turn, is passed from the department to the Health Visitors. It will be seen, therefore, that close liaison is established between the Chest Centre and the Health Department of the authority.

Respiratory tuberculosis is still more common in men than in women, and

there is a marked difference between the sexes in the age incidence of the disease. In females the incidence is highest between the ages of fifteen and thirty-five years, whereas in males it is highest between forty-five and sixty-five years. This higher incidence in the older age groups in men is probably influenced by the difference in the type of disease occurring in the sexes. Women between the ages of fifteen and thirty-five tend to have a more acute form of pulmonary tuberculosis than men, with resulting early death or cure. With men, the disease tends to follow a more chronic course in the same age group, and probably escapes detection in a large number of cases on this account. There is ample evidence to show that the disease in older males is due mainly to endogenous reinfection, and it is probable that the more chronic type of disease in young male adults tends to flare up in a majority of cases in middle or old age.

The death rate from pulmonary tuberculosis during 1950 was 22 per 100,000 of the population, and it is gratifying to record that during 1951 this rate was reduced to 17 per 100,000. The mortality rate in Derbyshire from this disease is probably one of the lowest in Britain. These rates reflect the incidence of disease being greatest in the younger age groups in women, and between the age group forty-five to sixty-five inclusive in men.

Northern Ireland Tuberculosis Authority.

The 6th Annual Report (1951) surveys a large number of subjects, including the Mass Radiography Service; a new Department to be established by the authority to deal with education, propaganda and research; Hospital Welfare Service; the medical examination of contacts; chemotherapy; and diversional therapy for domiciliary patients. The authority's plans for rehabilitation, including the employment of ex-patients within the service, the recruitment of home helps from quiescent cases of tuberculosis, and the staffing of the Multigraph Department by ex-patients, are also reviewed. For the future, a Rehabilitation Centre is visualised which will be linked to a 500-600 bed hospital.

With regard to the B.C.G. Vaccination Service, almost 6,000 vaccinations have been performed under the B.C.G. Scheme that has been in operation in a limited way since the month of July 1949. It was decided to widen this scheme to include new-born babies and school-leavers, and steps are now being taken to give effect to this decision. The wide application of the vaccine has called for the establishment of a special department to co-ordinate the arrangements in conjunction with Area Chest Physicians. The authority realises that if B.C.G. is to be widely applied within a relatively short space of time the active support and co-operation of the Northern Ireland Hospitals Authority and the County and County Borough Health Committees must be secured, and arrangements are about to be made to discuss with these bodies the best ways and means of carrying the scheme into Maternity Hospitals, Schools and Child Welfare Clinics.

Tuberculosis Control in Manitoba, 1951.

The Sanatorium Board of Manitoba is responsible for the following organisations and departments: X-ray surveys; Travelling Tuberculosis Clinics; Central Tuberculosis Clinic, Winnipeg; Manitoba Sanatorium, Ninette; Dynevor Indian Hospital, Selkirk; Brandon Sanatorium, Brandon; Clearwater Lake Sanatorium, The Pas; and co-operates with St. Boniface Sanatorium and King Edward Memorial Hospital. In his report for 1951, the Medical Director, Dr. E. L. Ross, explains that the objective of the Sanatorium Board is to reduce and ultimately eradicate tuberculosis from Manitoba, and the programme to

attain this end has been vigorously carried out during 1951. Broadly speaking, it is based on the discovery of new cases as early as possible, prompt and adequate treatment facilities, a sound and progressive rehabilitation service, and the co-ordination of all three. During 1951 in Manitoba 318,699 free chest X-rays were taken; 1,200 beds in seven institutions provided 416,265 days' treatment; and educational, vocational and rehabilitation services were provided for sanatorium patients.

Deaths have decreased this past year, the rate of 21·6 per 100,000 being the lowest ever recorded for Manitoba. The decrease in 1951 was due to fewer Indian deaths, which show a striking decline during the past four years. White population deaths were six more than the previous year, an increase of less than one per 100,000.

THE NATIONAL ASSOCIATION FOR THE PREVENTION OF TUBERCULOSIS IN AUSTRALIA

THE annual meeting of the Council of this Association, previously known as the Australian Tuberculosis Association, was held in Melbourne, Victoria, on August 22 and 23, 1952.

Delegates were present from the five affiliated State Divisions and the President, Dr. Keith Barry, took the chair. Among matters of interest reported or discussed were:

1. A statement by the President that the Governor-General of Australia, and the Governors of the five States, N.S.W., Victoria, South Australia, Western Australia and Tasmania, had all granted their patronage to the Association, as a Council of Honour.
2. Tentative plans towards the organisation of a Conference on Tuberculosis embracing Pacific and Eastern Asian nations, to be held possibly in Sydney in 1954.
3. The possibility of N.A.P.T.A.'s distributing a quarterly bulletin throughout Australia.
4. A Christmas seal design was selected for 1953.
5. A statement that all mail from the capital—and several other—cities, would be franked by the Post Office for two weeks in November with an appeal to buy Christmas Seals.
6. It was agreed that a Medical Section be formed, to be known as the Australian Laennec Society, and a suggested constitution for this Society was accepted by the Council.

The second annual general meeting of N.A.P.T.A. was held on the evening of Friday, August 22. After the disposal of business, members listened to a very entertaining and informative address by Sir John Storey, a prominent industrialist, the title being "The Influence of Sickness on the National Economy."

The Medical Section held a business meeting on the Saturday morning, August 23, when they agreed to submit their constitution to the Council of N.A.P.T.A. for its approval. They also elected office bearers for the ensuing year. As N.S.W. had already established a branch of the Laennec Society, and was the only State that had yet done so, it was agreed that the officers of the N.S.W. branch be appointed to similar offices of the society, namely: President, Dr. Cotter Harvey; President-Elect, Dr. Bruce White; Hon. Sec.-Treasurer, Dr. Bruce Geddes; N.S.W. Member, Dr. Gordon Bayliss.

NOTES AND NOTICES

INFLUENZA RESEARCH

Vaccine Trials to be Held this Winter

The Medical Research Council, in conjunction with the Ministry of Health, is organising trials of influenza vaccine this winter. About 15,000 volunteers—including a large number from industry, and others from among hospital nursing staffs, university students and similar groups—are taking part.

The trials started early in December 1952. Two vaccines are being used, one selected on the experience of last winter's pilot trials and the other similar in type to those which have been available for a number of years. Both are being produced in this country.

The following note gives the background to this winter's trials:

Two Types of Virus.—There are two types of influenza virus. The big epidemics which occur in this and other countries every few years have been due to virus A. The other type, due to virus B, occurs more frequently and is present more or less all the time. Virus A is the main cause of the high mortality and serious dislocation of industry which is a feature of influenza epidemics.

Although ordinary hygienic measures—good ventilation, avoidance of overcrowding and so on—may play a helpful part in reducing the incidence of virus influenza, the infection spreads so easily and so widely that immunisation of large numbers of the population would be advisable if a suitable means of vaccination were available.

In 1951, as the result of research into the virus A type of influenza, the matter was given special consideration by the Ministry of Health, and the Medical Research Council formed a Committee on Clinical Trials of Influenza Vaccines to carry on and co-ordinate the work. In the winter of 1951-2, with the aid of volunteer groups in the nursing staffs of hospitals and in the universities, some preliminary small-scale tests using specially prepared vaccines were completed. The results of these tests have put the Committee in a position to organise trials of a selected vaccine on a much larger scale in the winter of 1952-3.

The trials started early in December and it is hoped to include about 15,000 volunteers grouped in centres throughout the United Kingdom.

Two vaccines are being used, one selected and prepared as the result of last year's trials and—as a contrast—a vaccine similar in type to those which have been available for a number of years.

In each place where a trial is made half the volunteers are inoculated with one vaccine and half with the other. Special records of their health and illnesses are then kept for the following three months, and it is hoped that in the event of some of these groups being attacked by virus A influenza it will thus be possible to assess the preventive effects of the new vaccine as compared with the old.

THE THORACIC SOCIETY

The Spring Meeting will be held at the Royal College of Surgeons, Lincoln's Inn Fields, on Friday and Saturday, February 27 and 28, 1953. The Summer Meeting will be held in Oxford on Friday and Saturday, July 17 and 18, 1953.

MILFORD CHEST HOSPITAL

Retirement of Dr. R. J. Allison

Dr. R. J. Allison, on November 30, retired from the post of physician superintendent at Milford Chest Hospital. Nearly twenty-five years ago, when the hospital opened as the Surrey County Sanatorium, under the Surrey County Council, Dr. Allison was appointed as the first medical superintendent.

On Thursday, November 27, the staff and many past members of the staff and other visitors attended a reception at the hospital. Tributes to Dr. Allison were made on behalf of the South-west Metropolitan Regional Hospital Board by Sir Geoffrey Todd; on behalf of the Surrey County Council by Dr. K. A. Soutar, the County Medical Officer; by the chairman of the Hospital Management Committee, Mr. J. T. Pyne; and on behalf of the staff, both past and present, by Dr. A. S. Herington, deputy physician superintendent, who presented Dr. Allison with a Decca triple-speed record player, a cheque and an album containing the signatures of the contributors.

AMERICAN ASSOCIATION FOR THORACIC SURGERY

The thirty-third annual meeting of the American Association for Thoracic Surgery will be held on March 27, 28 and 30 in San Francisco, California.

FORMATION OF THE QUEBEC CHAPTER OF THE AMERICAN COLLEGE OF CHEST PHYSICIANS

At the joint meeting of La Société de Phthisiologie de Montréal et de Québec and the Tuberculosis Section of the Montreal Medico-Chirurgical Society held at the Sacred Heart Hospital, Cartierville, the President of the American College of Chest Physicians, Dr. Andrew L. Banyai, and the Executive Director, Mr. Murray Kornfeld, officially inaugurated the Quebec Chapter of the A.C.C.P., the first of its kind in Canada.

Dr. B. G. Bégin was elected Governor of the A.C.C.P. for the Province of Quebec; Dr. Fernand Hebert, Medical Director at the Sacred Heart Hospital, and Dr. J. J. Laurier, Assistant Director at the same hospital, were elected President and Secretary respectively.